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# **The growing horse: nutrition and prevention of growth disorders**



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## **The growing horse: nutrition and prevention of growth disorders**

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**European Workshop on Equine Nutrition**

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# **The growing horse: nutrition and prevention of growth disorders**

**EAAP publication No. 114**

## **Editors:**

V. Juliand and W. Martin-Rosset



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## Foreword

This book provides a compilation of papers presented at the 2<sup>nd</sup> European workshop on Equine Nutrition held at ENESAD in Dijon, France, January 15-17, 2004.

The 1<sup>st</sup> European workshop was initiated and organised in 2002 at ENESAD to promote exchanges between scientists and practitioners in Europe. It focused on the nutritional systems to meet the requirements of horses and was a great success. After these two pioneer workshops, it was decided to organise future meetings on a biannual basis under the umbrella of the EAAP. Publications of each of these meetings will be published in the EAAP scientific series. The overall scientific strategy of the forthcoming meetings is to study the physiology and nutrition of the different types of horses: Growing horses in 2004, mares in 2006, etc. Indeed, we need to determine the feeding standards for these different kinds of horses. There are very few books in the field which have been published recently to focus on three keys points: 1. The evaluation and prediction of nutrient requirements; 2. The means to overcome the limiting factors met in the application of the different nutritional systems for rationing horses, comparing the feeding practices throughout Europe, and; 3. The increasing interactions between health and nutrition, which limit the performance and the welfare of horses.

The workshop held in 2004 dealt with the growing horse, as this type of horse is quite representative of the situation designed previously in Equine physiology and nutrition. The growth period extends in horses between 5 to 7 years (including the period of pregnancy) e.g. 35 to 55 p100 of the productive life of a race, sport or leisure horse. This growth period requires a heavy technical and financial input for breeders and riders. This determines the further performances and longevity of the horse and the financial return for the horse producer and/or owner. The improvement of the knowledge in biology of growth, nutrition and physiopathology of the young horse is of major concern to communicate relevant husbandry and feeding systems to the horse industry. This could prevent the occurrence of costly growth disorders. The consideration of successful field practices could also significantly contribute to research and proposals issued from these research.

The scientific programme was determined by an international Scientific Committee consisting of the following members:

|                       |                 |
|-----------------------|-----------------|
| Dag Austbo            | Norway          |
| Domenico Bergero      | Italy           |
| Manfred Coenen        | Germany         |
| Derek Cuddeford       | Scotland        |
| Andrea Ellis          | The Netherlands |
| Pat Harris            | United Kingdom  |
| Michel Huard*         | France          |
| Véronique Julliand    | France          |
| Ellen Kienzle         | Germany         |
| William Martin-Rosset | France          |
| Nicoletta Miraglia    | Italy           |
| Yves Tazé*            | France          |

This workshop was organised by the Research Unit "Nutrition et santé des Herbivores at ENESAD (Etablissement National d'Enseignement Supérieur Agronomique) in Dijon, Burgundy county in France. Erwann Couedel was in charge of the secretariat.

The workshop was strongly supported by the club of French horse feed companies, the so called CNEF (Club de Nutrition Equine Français). The workshop was devoted to researchers, veterinarians, consultants, and skilled breeders and trainers. Hence, two representatives\* of CNEF were in the scientific committee to contribute to the elaboration of the scientific programme and especially the discussions and excursions.

The scientific programme was based around four main topics, organised in four sessions, concluded either by a round table, a poster session or by a general discussion, and two technical half day excursions:

- Session 1: Growth and development of the growing horse  
Poster session: growth curves and models  
Round table on the consequences of the new knowledge in growth and development and the need for further research;
- Session 2: Energy and protein requirements for growth  
General discussion on the nutritional systems used and the consequences of rationing and feeding practices;
- Session 3: Mineral and vitamin requirements for growth  
Poster session: role of the mare in nutrient supply  
General discussion on the scientific knowledge and consequences for evaluating the requirements;
- Session 4: Role of nutrition on developmental diseases  
Round table on the nutritional impact on the prevention of the developmental diseases.
- Excursion 1: National stud of Cluny (Haras nationaux)  
Meeting with breeders of AQPS (non-thoroughbred racing horse);
- Excursion 2: Selle français (a French breed of sport horses) stud farm (Haras de Vulsain)  
Auxois (a French draught breed) stud farm (Elevage de Vervres)

According to the format stated in the previous workshop, each session or visit was lead by a chairperson and the round table, general discussion, poster session or technical visit by a leader:

- Session 1: L.B Jeffcott and J.P. Valette
- Session 2: M. Coenen and Nicoletta Miraglia
- Session 3: E.A Ott and D. Cuddeford
- Session 4: Véronique Julliand and R. van Weeren

A discussion focused on husbandry and feeding practice in AQPS stud farms between breeders, practitioners and scientists was co-managed by D. Wuillaume (Haras nationaux) and Y. Taze (CNEF) in the scope of the National stud of Cluny, a city in the neighbourhood of Dijon.

Over 200 people from 20 countries attended the 3 days meeting. The meeting was financially supported by the horse industry (CNEF, WALTHAM company), the county of Burgundy and the city of Dijon, whereas the logistics were supplied by ENESAD, the agricultural organisation of Burgundy county, the National Stud (so called Haras nationaux), the breeder's unions of AQPS and Auxois, and finally by the Stud farm of Vulsain.

Veronique Julliand  
Head of the organising team in Dijon

William Martin-Rosset  
President of Horse commission at EAAP

**Part A**  
**Growth and development**





# Growth and development in the equine

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## Abstract

The horse seems to be an early animal. Body weight (BW) and Body Size (BS) reach: 9-10p100, 45p100 and 75p100 of adult BW at birth, one and two years respectively. Hence Average Daily Gain (ADG) is very high during suckling period then it decreases quickly after weaning. Most of BS parameters rise very quickly. Expressed relatively to adult size, height at withers reach 90p100 at one year; length and width 85-90p100 at two years. And BS is strongly linked to BW variation. Bone development is very early which is consistent with early BS evolution. Muscles content is very high (68-72p100). But adipose tissue development is late and the most variable. Body tissue and organs development is quite consistent with Hammond's pattern stated in farm animals. There is a strong development wave of bone and muscle from the distal to the proximal regions of the limbs and the trunk. BW and BS range from 1 to 5 according to breeds. ADG is strongly linked to adult BW. But the effect of breed on ADG and body composition intra group of breeds: heavy or light is limited. BW, BS and fat content of the body are higher in male than in female. But the effect of castration is limited in the male. BW, BS and body composition are subjected to the influence of the feeding level as much as BW at weaning is low. Horse is capable to compensate more or less the effect of restricted feeding. There is a strong linkage between the intensity of compensatory growth and the intensity of the restriction (feeding level x duration). But capacity for compensatory growth decreases with age. Adipose tissue development and content are highly subjected to nutrients intake and bone tissue to some extend too in interaction with physical exercise for the latest.

*Keywords: equine, growth, development, process, animal and environmental effects*

## Introduction

The growth period extends in horse between 5 to 7 years (including the pregnancy period) e.g. 35 to 55 p100 of the productive life for a race, sport or leisure horse. Thus the knowledge in the biology of the growth and development is of major concern for producing well developed and healthy horses.

The biology of growth and development is a very large area which extends from the implementation of the different tissues in the embryo to that of their morphological development during the life; and from the differentiation of the different types of cells to their organisation for ensuring a define function.

Our knowledge on growth and development in horse is based on work conducted at the end of the 19<sup>th</sup> or at the beginning of the 20<sup>th</sup> centuries in Western Europe, or in the fifties in the Eastern Europe, and since the sixties in USA and again in Western Europe due the rising interest in sport and leisure horses in the industrialised communities. The studies conducted in the very early time were mainly focused on the description of body size and to a lesser extend

in body weight and their variation with age or difference with breeds. The work carried out during the first half-part of the 20<sup>th</sup> century was dedicated mainly to study the variation of body weight with age and the influence of the sex, the breeds, the parity of the mare and the date of birth. In the most recent studies, the influence of nutrients intake on the body weight was of major concern. But the knowledge on body composition in the horse was significantly increased thanks to the work carried out in the seventies in France, and in Italy to a lesser extend, in the heavy horse dedicated to meat production and thus slaughtered at different ages of the growth period to be anatomically dissected as in other farm animals.

The aims of this review are:

- to highlight the majors figures of the evolution of body weight, body size and body composition;
- to determine the influence of the major factors of variation such as: breed, sex, nutrition.

There is no extended comparison with other animals which has been done.

## Body growth

### Definition

From the fertilization until the adult age, body weight (BW) of horse increases with time (t), and  $BW = f(t)$

### Curve and general model

In horse BW increases since fertilization to adult stage according to a sigmoid curve (Figure 1). And growth has been described using different equations.

The most simple curve and model were proposed by Brody 1945 According to this curve two periods are designed: an accelerated period ( $P_1$ ) then a delayed period ( $P_2$ ) (Figure 1a). During the period  $P_1$ , the instantaneous growth rate increase with the time  $\frac{dBW}{dt}$  -

As far as the number of cells rises, the growth rate is proportional to body weight (BW) of the horse (Figure 1c):  $\frac{dBW}{dt} = KBW$  (1)

eq1 becomes  $BW = f(t)$  after mathematical integration and it comes:

$$BW = BW_0 e^{kt} \quad (2)$$

$BW_0$  = Body Weight at birth (Figure 1a)

K can be calculated from  $\log BW = \log BW_0 + Kt$

During the period  $P_2$ , growth rate decreases with the time as it is affected by different inhibiting factors: amount of nutrients, enzymes.... which are available... etc.  $\frac{dBW}{dt}$  is proportional to the difference between adult  $BW_A$  and BW at a present time (Figure 1c).

$$\frac{dBW}{dt} = K'(BW_A - BW) \quad (3)$$

eq3 becomes  $BW = f(t)$  after mathematical integration and it comes:

$$BW = BW_A - be^{-k't} \quad (4)$$

$BW_A$  = Adult Body Weight

b is a constant.

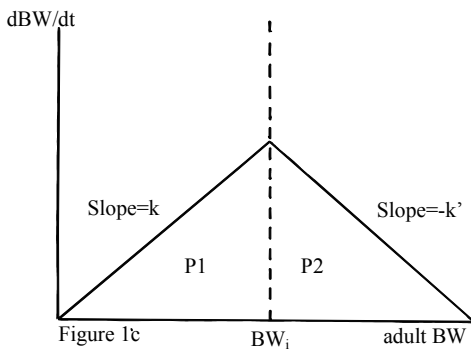
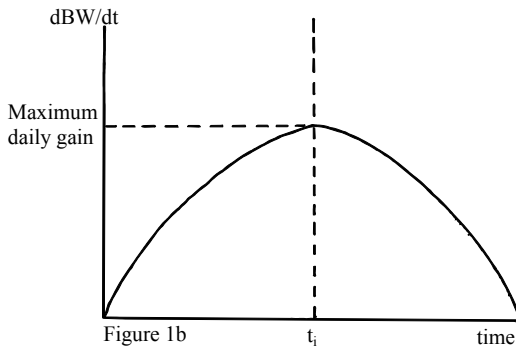
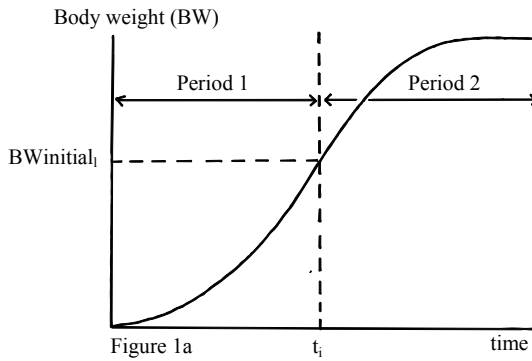


Figure 1. Growth curve according to Brody (1945).

At the point of inflection:  $BW_i$ ;  $t_i$ , growth rate is at a maximum (Figure 1b). It is supposed that the horse reaches puberty at this point. In most mammals  $BW_i$  accounts for 30 p.100 of  $BW_A$ , except in human where  $BW_i = 60-70$  p.100  $BW_A$ . Age at  $t_i$  is a physiological reference which is not well defined in horse.  $BW_A$ ,  $BW_O$ ,  $b$ ,  $k$ ,  $k'$  and  $BW_i$  are physiological characteristics for a species, a breed or an individual animal. As a result  $BW_A$  cannot be overcome as it is determined by genetic potential, and growth potential of horse is directly linked to  $BW_A$ . But  $BW_A$  can be reduced as far as the horse is negatively affected by external factors, namely nutrition.

Others equations have been proposed to describe growth:

$$\text{Equation of Gompertz (Winsor 1932): } \log BW = e^{(A+bRt)} \quad (5)$$

$$\text{Equation logistic (Pearl and Reed 1923): } BW = \frac{1}{A + bRt} \quad (6)$$

$$\text{Equation of Richards 1959: } BW = A (1 - be^{-Kt})^M \quad (7)$$

The biological interpretation of such equation has been discussed extensively by Fitzhugh 1976

A = asymptotic limit for BW as BW reaches infinity

b = adjustment for initial BW

$$k = f\left(\frac{\text{maximum growth rate}}{BW_A}\right)$$

k could be used either to determine growth rate or rate of change in growth rate.

M is the point of inflection in the curve (Figure 1)

ADG: growth rate is estimated to be the first derivative of eq 7.

BWM: is the percent maturity

$$BWM = (1 - e^{-Ke})^M$$

### Body weight at birth and foetal growth

Body weight at birth ranges from 23 to 77 kg according to the breeds. Body Weight represents 7 to 13 p.100 of adult BW (Table 1). BW at birth ( $BW_f$ ) can be predicted from BW of the mare: BW<sub>m</sub> using the following model:  $BW_f = 0.45 BW_m^{0.75}$ . Until the seventh month of pregnancy cellular multiplication is very high according to this general model (Figure 2).

$$BW = Bow e^{Kt}$$

Then body mass rises very quickly according to the general model stated by INRA 1984 from the data of Douglas and Ginther (1975) in ponies; Dusek 1966, Meyer and Ahlswede (1976) in the light breeds and Martin-Rosset unpublished in heavy breeds (Figure 2).

$$Y = -a + bx - cx^2 + dx^3 - ex^4$$

$$Y = \frac{BW_f \text{ (foetus)}}{BW_b \text{ at birth}}$$

$$BW_b = \begin{cases} 17 \text{ kg for Ponies} \\ 53 \text{ kg for light breeds} \\ 67 \text{ kg for heavy breeds} \end{cases}$$

$$X = \frac{\text{number of days at a determined date from fecondation}}{\text{duration of pregnancy}}$$

Meyer and Ahlswede (1976) proposed a linear equation, too.

Table 1. Body weight at birth and at adult age (drawn from Langlois, 1973; Platt, 1984).

|                  | Adult body weight (1)<br>(kg) | Body weight at birth (2)<br>(kg) | (2) / (1)<br>(%) |
|------------------|-------------------------------|----------------------------------|------------------|
| Shire            | 1016-1118                     | 68-77                            | 6.8              |
| Thoroughbred     | 505                           | 50.3+/-5.9                       | 10.0             |
| Lippizan         | 408-460                       | 42.5                             | 9.8              |
| Pure-bred Arab   | 418-432                       | 41.5                             | 9.8              |
| Shetland         | 173                           | 23.0                             | 13.3             |
| Przewalski horse | 250-325                       | 25-30                            | 9.6              |
| Zebra            | 340                           | 30-35                            | 9.6              |
| Saddle horse     | 565                           | 53+/-4                           | 9.5              |

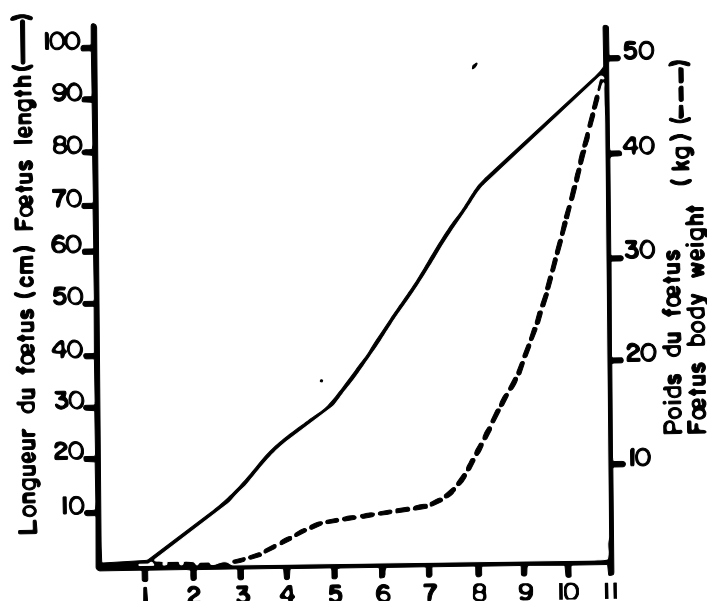


Figure 2. Variation of body weight and length of the foetus during the pregnancy (drawn from Dusek 1966).

At birth, BW of colt would be 2.5 to 3.5kg higher than that of fillies in Belgian heavy breed (Butaye, 1966). But this discrepancy could be due to a longer duration of pregnancy: +1.7 day (Mauch, 1933; Howell and Rowllins, 1951; Zwolinski, 1966; Ropiha *et al.*, 1969; Joubert *et al.*, 1969).

BW at birth would be 2 to 7 kg lower in progeny born from primiparous than from multiparous mare (Butaye, 1966; Martin-Rosset and Trillaud-Geyl, 1984). BW at birth would be maximum in progeny born from 6-7 years old mares (Ilancic, 1956).

The date of birth would influence BW at foaling, but this influence is confusing with the duration of pregnancy as this later would explain 43p100 of the total variation of BW (Howell and Rollins, 1951). The duration of pregnancy can be 6 to 10 days shorter as far as the foaling

take places in autumn or in spring. As a result BW at birth of horse of light breeds would be higher in spring than in autumn (Jordao and De Camargo, 1950; Lohman and Marinic, 1952).

### Adult body weight (BW) and average daily gain (ADG)

Adult body weight range between 180 to 1100 kg according to the breeds (Table 1). Adult BW can be reached between 4 to 7 years. After birth BW rise curvilinearly until mature BW (Figure3). There is no loss of weight at birth in horses. BW is two times heavier at one month of age as milk production of the mare is rising. ADG range between 1600-2000 g in light or heavy breeds respectively (see review Martin-Rosset, 1983a). At 3 months of age ADG is 1200-1400 g in light and heavy breeds respectively as far as milk yield decreases and grass intake rises. At weaning BW is 5 times higher than at birth, it accounts for 45 p.100 of adult BW. ADG is still 1000-1200 g in light and heavy breeds respectively. BW reaches: 65, 72 and 95 p.100 of adult BW at 18, 24 and 42 months respectively as ADG decreases subsequently from 800-1400 g, then 400-700, to 0-100 g at respective ages in high or heavy breeds respectively (Figure 3).

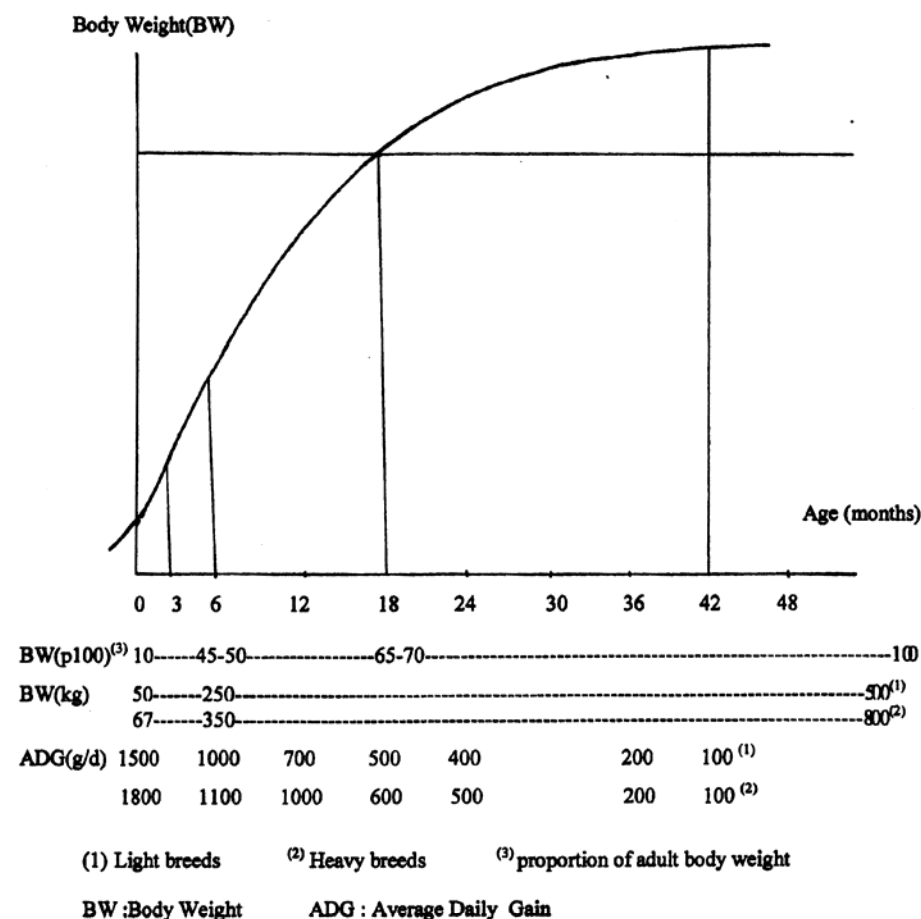


Figure 3. Variation of body weight in horses from birth to adult age.

## Body size

### Definition

The variations of body regions, organs and tissues with age are different respectively. As a result size and body composition change according to specific patterns. All these variations describe the development in the horse.

### Methodologies

The variations in body size have been extensively determined using different methods: Lydin gauge, tape measure...., or photography at a constant scale (see review of Martin-Rosset, 1983a).

### Body size variation with age

During the pregnancy the length and height at withers rise strongly since the 5-7<sup>th</sup> months respectively (Figure 2) as far as the length of the limb increases (Köenig, 1960; Green, 1969): 60-70 p.100 to the value reach at birth. At birth, height at withers account for 60p.100 of adult value (Dusek, 1972) versus 57p.100 in cattle (Beranger, unpublished). Comparing to cattle, horse seems to be early mature animals.

From birth to adult age body size variation follows a pattern which has been drawn from the studies of early workers (see review of Martin-Rosset, 1983a). During the first postnatal year, the body size of the foal can be included into a standing up rectangular and height at withers reach at 12 months already 90 p.100 of adult value (Figures 4a and b). Then, the length and the width increase. At 18 months, the body size can be included into a square. At 2 years of age, the trunk length and thoracic perimeter account for 95 and 90 p.100 of adult value. At the final stage, the body size can be included into a lying rectangular. Adult size is met between 4 to 7 years old according to the breeds.

The size is strongly related to BW as pointed out by the different models performed to predict BW from various parameters of body size (Table 2).

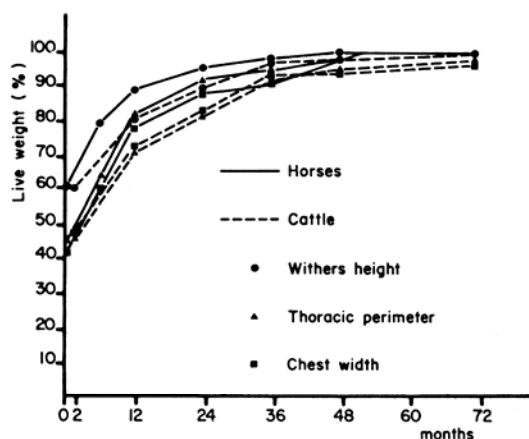


Figure 4a. Evolution of body size after birth in horses (from Dusek 1972).

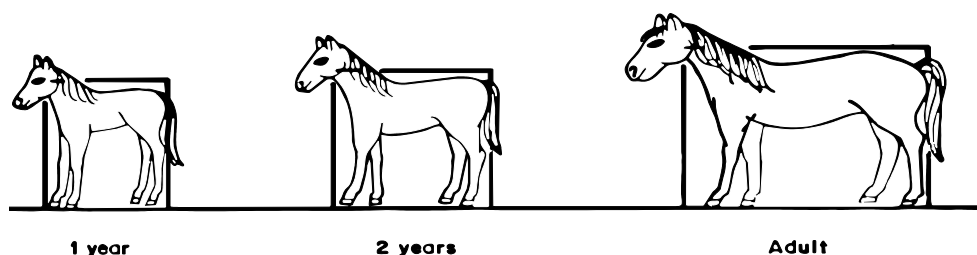


Figure 4b. Evolution of body size after birth in horses (drawn from review of Martin-Rosset 1983a).

Table 2. Prediction of body weight from body size in horses.

| Authors                | Equations                                                                                                | Breeds | Type                                                        |
|------------------------|----------------------------------------------------------------------------------------------------------|--------|-------------------------------------------------------------|
| INRA 1990 <sup>1</sup> | $BW = 4.5 TP - 370 \pm 23$<br>$BW = 5.2 TP + 2.6 HW - 855 \pm 25$<br>$BW = 4.3 TP + 3.0 HW - 785 \pm 26$ | Light  | Growing horse (6 - 48 month)<br>Mare<br>Stallions, Geldings |
| ENVA 2000 <sup>2</sup> | $BW = 0.204 \text{ Age} + 1.893 \text{ HG} +$<br>$2.013 TP - 333.9 \pm 16$                               | Light  | Growing horses (0-24 months)                                |
| INRA 1990              | $BW = 7.3 TP - 800 \pm 27$                                                                               | Heavy  | Growing horses                                              |

BW = Body weight (kg)

TP = Thoracic Perimeter (cm)

HW = Height at withers (cm)

<sup>1</sup> Bigot *et al.*, 1988

<sup>2</sup> Paragon *et al.*, 2000

## Body composition

### Methodology and allometry

The ponderal variations of body composition are determined using the comparative slaughter method when the animals are slaughtered sequentially at subsequent ages in the scope of an experiment designed for that purpose. The different tissues are anatomically dissected after slaughter and weighted; the organs too. Such a method is very heavy and quite expensive. It has been scarcely implemented in horses (Gunn, 1975; Martin-Rosset *et al.*, 1983b).

The variation of the weight of tissues or organs are analysed using the allometric equation of Huxley, 1932:  $y = a x^b$ . In this equation  $b$  is the allometric coefficient of  $y$  relatively to  $x$  ( $b = \frac{y}{x}$ ):  $b$  represent the relative growth of a body part  $Y$  to a reference  $X$  which could be empty body weight by example. If  $b$  is higher than 1, this part of the body is having a faster relative growth than that of the reference part and conversely. And if  $b=1$  his part is following an isometric development with the reference part.



## Variation in body composition with body weight

The ponderal variations of body regions, tissues and organs were described in heavy breed slaughtered and then anatomically dissected between birth to 3 years (Martin-Rosset *et al.*, 1979 and 1983b).

The relative growth of most of the *organs* range between  $b = 0.5$  and  $b = 0.8$  except for the large intestine where  $b = 1.31$ , (and the stomach to a lesser extend  $b = 1.15$ ) as the horse is getting to be an herbivore mainly after 12 months of age (Table 3).

Table 3. Relative growth of various components of the whole empty body between birth to 30 months in horses (drawn from Martin-Rosset *et al.*, 1979 and 1983b).

|                                       | bo $\pm$ Sb     | RVC <sup>1</sup> p.100 |
|---------------------------------------|-----------------|------------------------|
| Carcass                               | 1.07 $\pm$ 0.01 | 2.6                    |
| Carcass components                    |                 |                        |
| Fatty tissues                         | 1.45 $\pm$ 0.08 | 42.9                   |
| Muscles                               | 1.13 $\pm$ 0.01 | 5.1                    |
| Bone                                  | 0.74 $\pm$ 0.02 | 9.6                    |
| 5 <sup>th</sup> quarter               |                 |                        |
| Blood                                 | 0.63 $\pm$ 0.06 | 16.1                   |
| Spleen                                | 0.82 $\pm$ 0.03 | 17.4                   |
| Hearth                                | 0.78 $\pm$ 0.02 | 9.9                    |
| Kidney                                | 0.61 $\pm$ 0.03 | 15.8                   |
| Lungs                                 | 0.54 $\pm$ 0.03 | 17.0                   |
| Liver                                 | 0.53 $\pm$ 0.02 | 11.8                   |
| Digestive tract                       |                 |                        |
| Stomach                               | 1.15 $\pm$ 0.05 | 26.0                   |
| Small intestine                       | 0.79 $\pm$ 0.05 | 28.3                   |
| Large intestine                       | 1.31 $\pm$ 0.05 | 23.9                   |
| 5 <sup>th</sup> quarter fatty tissues | 1.20 $\pm$ 0.07 | 40.2                   |
| Hide                                  | 0.85 $\pm$ 0.02 | 10.4                   |
| Head                                  | 0.77 $\pm$ 0.03 | 9.2                    |
| Total fatty tissues                   | 1.41 $\pm$ 0.07 | 40.3                   |

<sup>1</sup>Residual variation coefficient (standard error of y expressed in p.100 of y)

As far as *skeleton* is concerned, the relative growth of the fore-and hind- limbs is close to 1. A gradient can be noticed from the metacarpus or metatarsus to the scapula or the pelvis, where  $b$  is much lower or higher than 1 respectively. In the trunk, relative growth of vertebra and ribs is much higher than 1 (Figure 5). The relative growth of the different regions of the skeleton is consistent with the body size evolution.

The muscles can be divided in 4 regions according to their relative growth. The distal part of the limb ( $b = 0.80$ ), the neck and the shoulder ( $b = 0.93$ ), the thoracic and dorsal region and the tight ( $b = 1.04$ ) and the abdominal region ( $b = 1.07$ ) (Figure 6). However, no great

variation in the proportion of the different muscular region was observed with the exception of the abdominal region and the fore limbs (Figure 6).

The relative growth of total *adipose tissues* is very high and variable ( $b = 1.41$ ; Residual Coefficient Variation = 40.3 p100). The development of adipose tissue in the 5<sup>th</sup> quarter is shortly earlier ( $b = 0.8$ ) than in the intermuscular adipose tissue ( $b = 0.9$ ) or in the internal carcass and subcutaneous tissue ( $b=1.2$ ).

The pattern of the relative growths of the organs, tissues and regions are similar in the horse as that described by Hammond, 1932 in the other mammals (Table 4).

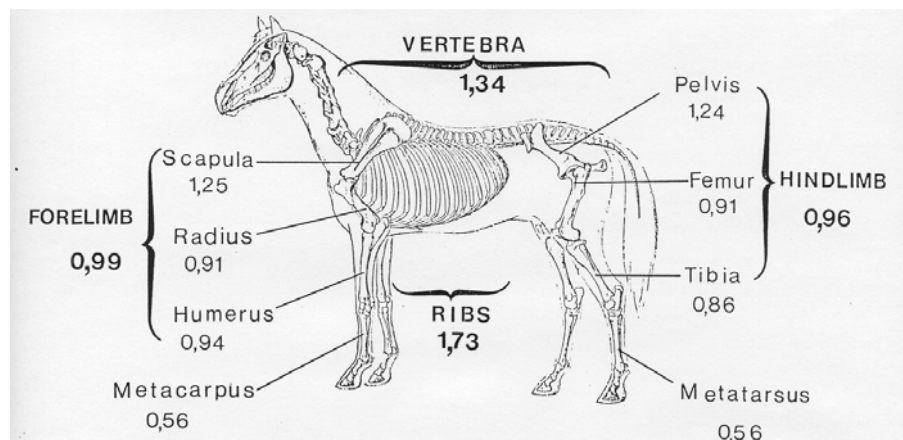


Figure 5. Relative growth of skeleton in the horse between birth and 30 months (from Martin-Rosset et al., 1979).

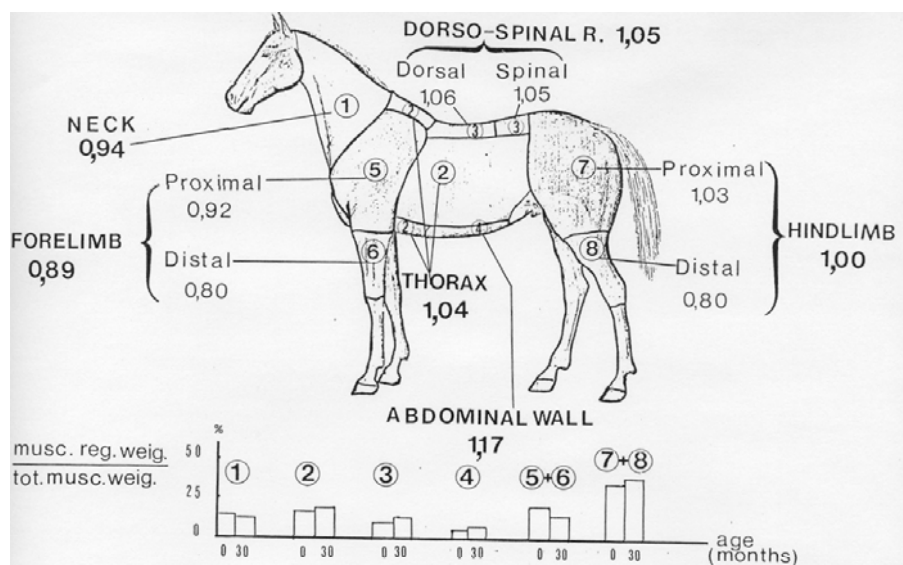


Figure 6. Relative growth of different muscular regions in the horse between birth and 30 months (from Martin-Rosset et al., 1979).

Table 4. Relative growth pattern of Hammond 1932.

|            | Curve 1  | Curve 2       | Curve 3      | Curve 4       |
|------------|----------|---------------|--------------|---------------|
| Tissue     | Nervous  | Bone          | Muscular     | Adipose       |
| Regions    | Head     | Neck          | Trunk        | Croup         |
| Limbs      | Cannon   | Tibia Humerus | Femur Radius | Pelvis        |
| Fat tissue | Internal | Intermuscular | Subcutaneous | Intramuscular |

### Consequences of the variation in body composition with body weight

In the carcass, the proportion of skeleton decrease from 31 to 14 p.100, whereas the proportion of muscles and adipose tissues increase from 59 to 72 p.100 and 6 to 14 p.100 respectively between 12 and 30 months of age (Martin-Rosset *et al.*, 1983b).

Adipose tissues of the carcass average 88 p.100 of total adipose tissues but they increase from 86 to 89 p.100 between 12 and 30 months of age. As a result, the distribution of adipose tissues changes (Figure 7). Internal adipose tissues increase strongly whereas inter muscular

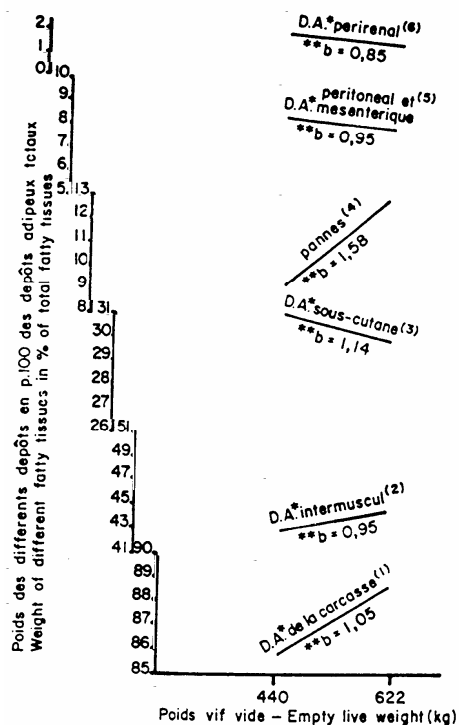


Figure 7. Distribution of fatty tissues according to empty live weight. Relative growth of various fatty tissues (Y in kg) as compared to total fatty tissues (X in kg) (from Martin-Rosset *et al.*, 1983b).

\*D.A.: Fatty tissues

\*\*b: Allometric coefficient

<sup>1</sup> Carcass, RVC=3.5 p. 100.

<sup>2</sup> Intermuscular RVC=17.1 p.100.

<sup>3</sup> Subcutaneous RVC=20.4 p.100.

<sup>4</sup> Thoracic fat RVC=28.5 p.100.

<sup>5</sup> Peritoneal and omental RVC=34.2 p.100.

<sup>6</sup> Kidneys RVC=63.8 p.100.

RVC=Residual variation coefficient

adipose tissues rise slightly. It should mention that the proportion of intermuscular adipose tissue is only 43 p.100 in horses compared to 56 p.100 in cattle (Beranger and Robelin, 1977). Between 440 to 622 kg EBW, the relative variation of the proportion of *lipids* increases from 40 to 50 p100 in the Empty Body Weight (EBW)(and 233p100 between 283 and 622 kg) (Figure 8). Lipids content is linked to body mass according to an allometric equation:

$$\text{Lipids} = 0.03 \text{ EBW}^{1.6} \quad (8)$$

similarly to that observed in cattle (Robelin and Geay, 1978).

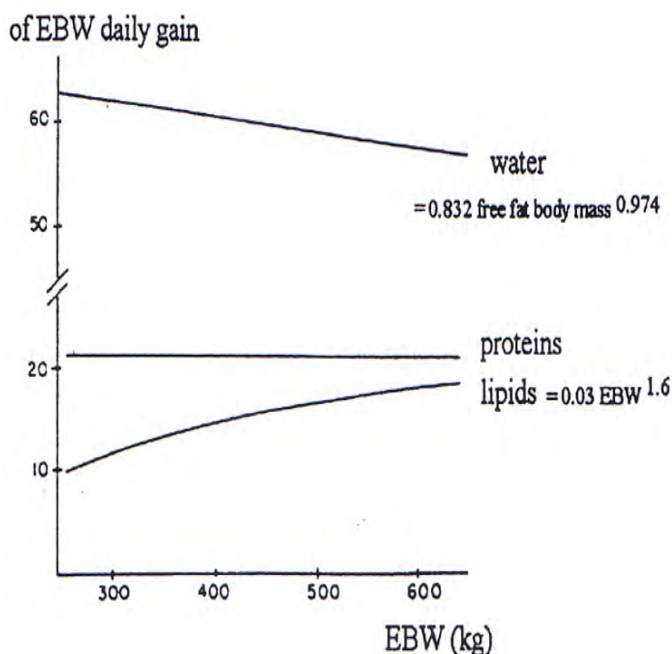


Figure 8. Variation of chemical composition of body during daily gain for growing horses (source: Agabriel et al., 1984).  
EBW: Empty Body Weight

But *water* content decreases too from 75 p.100 to 70 p.100 between birth and adult age (Meyer and Ahlswede, 1976; Robb *et al.*, 1972). As a result there is a relationship between Free Fatty Mass (FFM=EBW-lipids or water + proteins + ash) and Water Mass (WM) in the horse similar to that observed in cattle (Robelin and Geay, 1978).

$$\text{WM} = 0.832 \text{ FFM}^{0.974} \quad (9)$$

The protein content of dry FFM averages 78 p.100 as it ranges between 77 and 79p100 (Robb *et al.*, 1972; Schryver *et al.*, 1974).

Using all these data and hypothesis, chemical composition of 1 kg EBW in lipids, proteins and water were calculated and described as far as EBW rises (Figure 8). 20.0 %, 11.5 % and 62.0 % of proteins, lipids and water are fixed respectively in 1 kg of EBWG at 300 kg EBW, whereas at 600 kg EBW 20 %, 18 % and 57 % of protein, lipids and water are deposited. It

shows that lipids content of EBW Gain increases whereas water content decreases and protein keeps on being steady as far as EBW rises in horses as in cattle.

But the chemical composition of EBWG varies with ADG the horses (Table 5). By example between 6 and 12 months lipids content of daily EBW gain is 3 times higher and protein content is 28 p.100 lower as far as ADG rises from 0.4 to 1.1 kg. But between 12 and 18 months of age, lipids content of daily EBW gain is 2 times higher and protein content is only 10 p.100 higher as far as ADG increases from 0.8 to 0.9 kg.

*Table 5. Chemical composition of daily gain in yearling and long yearling of breed in different situations (drawn from Agabriel et al., 1984).*

| Type of animal                     | Daily gain<br>of BW <sup>1</sup> | Daily gain<br>of EBW <sup>1</sup> | Chemical composition<br>of EBW <sup>1</sup> daily gain |                  | Chemical composition<br>of BW <sup>1</sup> daily gain |                  |                  |
|------------------------------------|----------------------------------|-----------------------------------|--------------------------------------------------------|------------------|-------------------------------------------------------|------------------|------------------|
|                                    | g/d                              | g/d                               | Lipids<br>p.100                                        | Protein<br>p.100 | Lipids<br>p.100                                       | Protein<br>p.100 | Energy<br>Cal.   |
| Yearling (6-12 months)             |                                  |                                   |                                                        |                  |                                                       |                  |                  |
| 1 <sup>st</sup> trial <sup>2</sup> | 1100                             | 890                               | 13                                                     | 20.9             | 10.6                                                  | 16.9             | 1890             |
| 2 <sup>nd</sup> trial <sup>3</sup> | 500                              | 400                               | -5.5                                                   | 25.2             | -4.4                                                  | 20.2             | 825 <sup>4</sup> |
|                                    | 1000                             | 800                               | 11.3                                                   | 21.1             | 9.0                                                   | 16.9             | 1720             |
|                                    | 1390                             | 1100                              | 15.9                                                   | 19.7             | 12.6                                                  | 15.6             | 2060             |
| Long yearling (12-18 months)       |                                  |                                   |                                                        |                  |                                                       |                  |                  |
| Growth during the prior winter     |                                  |                                   |                                                        |                  |                                                       |                  |                  |
| Slow                               |                                  | 950                               | 8 - 11                                                 | 21 - 22          |                                                       |                  |                  |
| Moderate                           |                                  | 840                               | 2 - 6                                                  | 23 - 24          |                                                       |                  |                  |

<sup>1</sup>BW = Body Weight; EBW = Empty Body Weight

<sup>2</sup>Average values observed in yearlings fed with the same diet: anatomical composition was measured by dissection of half carcass

<sup>3</sup>Referenced values obtained when comparing yearlings affected by different feeding levels: anatomical composition was predicted from anatomical dissection of the 14<sup>th</sup> rib according to Martin-Rosset *et al.*, 1985.

<sup>4</sup>Lipids content was considered to be 0

The development is quite complex as far as all this tissues are concerned. The understanding is simplified whereas the lean body mass (muscle + bone) or free fatty body mass (water + protein + ash) are concerned rather than body weight.

Composition of lean body mass is very close to that of the carcass and less variable (Table 6). Chemical composition of free fatty body mass is nearly steady 75-78 p.100 of water, 19-21 p.100 of protein and 4 p.100 of ash (Meyer and Ahlswede, 1976; Robb *et al.*, 1972; Schryver *et al.*, 1974). As a result free fatty body mass seems to increase without any large variation in chemical composition whereas there would be a fat deposition.

## Cellular mechanisms of growth

### Methodology

The growth of the tissues involves the variation in the number of cells (hyperplasy) and their size. If a cell is designed as a nucleus and the average amount of materials, the number of nucleus (N) gives the number of cell, which is associated. The size (or average weight) of cell can be estimated using the ratio  $p = \text{fresh weight of materials} / N$ . The weight of an organ or a tissue P is proportional to cell size:  $P = Np$ . As far as the amount of DNA is steady in the

nucleus for a species (Boivin and Vendrely law), the number of cells is proportional to the amount of DNA.

$$N = K (DNA)$$

$$\text{Cell size } p = \frac{P}{N} = \frac{P}{K(DNA)}$$

$$\text{As a result } P = K (DNA) p$$

$$\text{and } \frac{\Delta P}{P} = \Delta \frac{(DNA)}{(DNA)} + \frac{\Delta p}{p}$$

During an interval of time  $\Delta t$ , the relative variation of the weight of a tissue or an organ  $\frac{\Delta P}{P}$  comes from the sum of the relative variations of the cell numbers  $\frac{(\Delta DNA)}{DNA}$  and the cell size  $\frac{\Delta p}{p}$ .

Table 6. Variation of lean body mass with age in horses (drawn from Martin-Rosset et al., 1983b and unpublished).

| Age (months)               | Body weight (kg) | p.100 adult body weight | Carcass weight (kg) | Muscle/bone | Muscles <sup>1</sup> p.100 | Skeleton <sup>1</sup> p.100 |
|----------------------------|------------------|-------------------------|---------------------|-------------|----------------------------|-----------------------------|
| Foetus (8mo.) <sup>1</sup> | 23.5             | 3.1                     | 13.2                | 1.41        | 57.3                       | 41.1                        |
| 0 <sup>2</sup>             | 61.5             | 8.1                     | 37.4                | 1.88        | 59.0                       | 31.7                        |
| 6 <sup>3</sup>             | 319.0            | 40.1                    | 186.4               | 3.87        | 69.8                       | 18.4                        |
| 12 <sup>4</sup>            | 483.2            | 59.2                    | 313.4               | 4.48        | 70.1                       | 15.6                        |
| 18 <sup>5</sup>            | 572.8            | 70.1                    | 329.0               | 4.46        | 71.8                       | 16.1                        |
| 24 <sup>6</sup>            | 626.8            | 76.9                    | 382.7               | 4.69        | 69.8                       | 14.9                        |
| 30 <sup>7</sup>            | 735.3            | 90.1                    | 440.9               | 4.81        | 69.0                       | 14.5                        |

Number of animals: 4<sup>1</sup> – 4<sup>2</sup> – 13<sup>3</sup> – 20<sup>4</sup> – 20<sup>5</sup> – 20<sup>6</sup> – 15<sup>7</sup>

<sup>1</sup> p100 in the carcass

The relative growth can be determined measuring the variation in DNA and of the fresh material/DNA ratio, and the relative effect of the increase in the number or the size of cells on the growth of tissue or an organ can be estimated.

The protein synthesis is the major factor in growth. RNA monitors this synthesis. The variation in protein synthesis is estimated by determining the amount of proteins (proteins/DNA).

### Cellular activity

Drawn from data obtained in other mammals (rat, pig...) the growth curve meets a point of inflection at the puberty. After weaning the cell number (N) in the carcass is described by a hyperbolic function of its weight P:

$$N = \frac{aP}{bP + C} \quad (a, b, c, \text{ are constants})$$

The growth of the carcass comes from the cellular multiplication (hyperplasia) until the puberty then the increase in cell size (hypertrophia) become of major concern, but cellular multiplication is not closed (Figure 9).

$$N \text{ (cell numbers)} = aW/(bW+c)$$

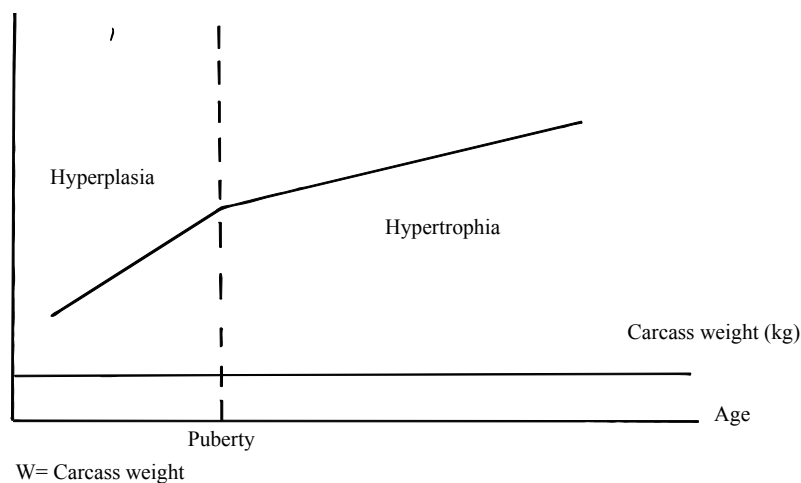


Figure 9. Cellular mechanisms of growth in mammals.

## Osteogenesis

During the bone growth, the bone tissue is affected by modelling and remodelling. Modelling is more important in the growing horse as a result there is the formation of a bone tissue (Maenpaa *et al.*, 1988) whereas remodelling occurs mainly in the adult horse. The bone tissue is a conjunctive tissue with cells and intercellular materials, which is calcified during the growth process. The osteoblasts are involved in the bone production. The bone organic matrix is elaborated by the osteoblasts. And the calcification is monitored by the osteoblasts too (Holtrop, 1975; Franck, 1979). The bone tissue is resorbed by the osteoclasts (Baron *et al.*, 1986). The osteocytes are cells, which belong to the osteoblastic line, and they are capable either of synthesis or resorption. The intercellular substance is an organic matrix of collagen fibrils mainly where minerals are deposited. The architecture of bone tissue changes with age. The osteogenesis involves:

- the division of fusiform cells of the primary mesenchyme which elaborates a fundamental substance with a high content in collagen and proteoglycans;
- the condensation of cells which produce a preliminary bone;
- the differentiation of fusiform cells in chondrocytes (and their multiplication) which synthesise the cartilaginous matrix and contribute to the increase in size of long bone;
- and from the transformation of the cartilaginous matrix in final bone so-called endochondral ossification.

The ossification process of the preliminary bone occurs in three phases. The primary ossification which produces a fibrous bone. Calcified cartilaginous matrix (or septas) is elaborated by the hyperplasia of the chondrocytes. These fibrous spans are produced by the dual action of osteoclasts and osteoblasts. During the subsequent secondary ossification, the non-lamella fibrous bone, which is characterised by non oriented collagen fibrils, is substituted by lamella bone tissue. The growth in length occurs as far the growth plate is active. Serriated cartilage is elaborated by proliferative chondrocytes (Figure 10). Those cells produce cartilaginous matrix, which is then substituted by bone tissue according to the endochondral

ossification process (Figure 11). The transformation of the cartilaginous matrix into the lamella bone tissue is achieved at the puberty (18-30 months according to the sex). The metaphyseal growth plate is strongly involved in the process (Figure 11). But the production of bone tissue is decreasing quickly after birth as described by the curve of osteocalcine concentration in the plasma of the foal (Figure 12).

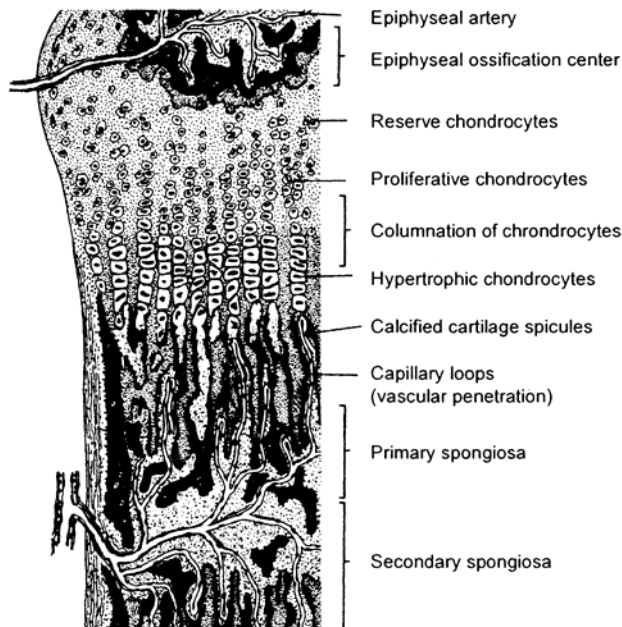


Figure 10. Anatomy of metaphyseal growth plate (adapted from C.W. McIlhwaith, 1998).

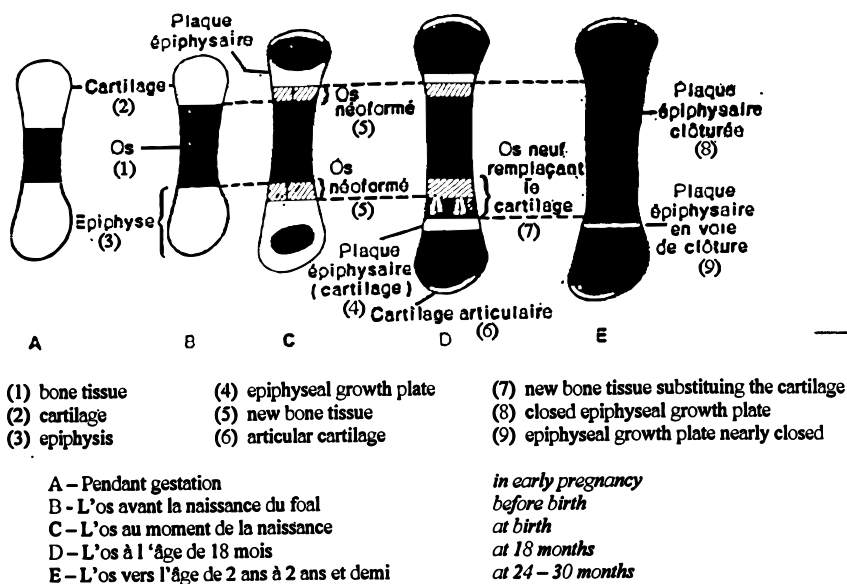


Figure 11. Bone growth of long bone.



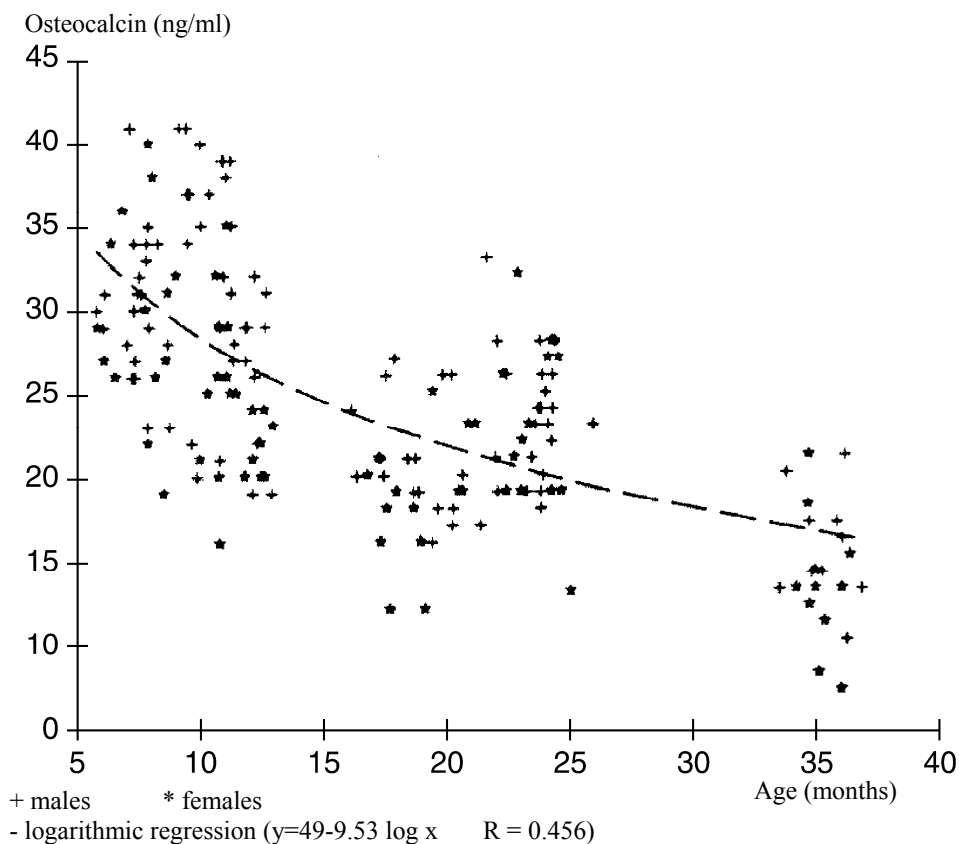


Figure 12. Variation of osteocalcin (Bone Gla Protein or BGP) in plasma with age and sex (Bigot *et al.*, unpublished, quoted in Martin-Rosset, 2001).

### Consequences of osteogenesis and allometry of bone tissue on mechanical properties of bone: the cannon bone

The mechanical properties of the cannon bone were extensively studied at INRA using the 4-point bending test in respect of its physico-chemical characteristics (Bigot *et al.*, 1990 and 1996). Between birth and 40 months of age, the increase in the length accounts for only 20 p.100 of adult size. But the weight, the volume and the thickness are 2 times higher whereas the bone density is only 20 p.100 higher (Figure 13a). The thickness of diaphysis, namely cortical bone, which is 2 to 3 times higher, is responsible of the increase in weight and volume (Figures 13a and 13b). The mineral content does not change significantly in respect of the age and body weight. The breaking stress ( $S$ : maximal strength/unit of surface before breaking) and the axial rigidity or modulus of elasticity ( $E = \Delta S$ ) increase exponentially in respect of age and body weight (Figure 14a and 14b). Conversely the ultimate specific deflection:  $Eu = D$  (deformation before breaking)/ $L$  (distance between the 2 support-points for determining  $Eu$ ) decreases very early in respect of age and body weight (Figure 15). The cortical bone would meet the greatest proportion of its mechanical properties at weaning. But this variation is depending of sex, nutrient allowances and exercise too.

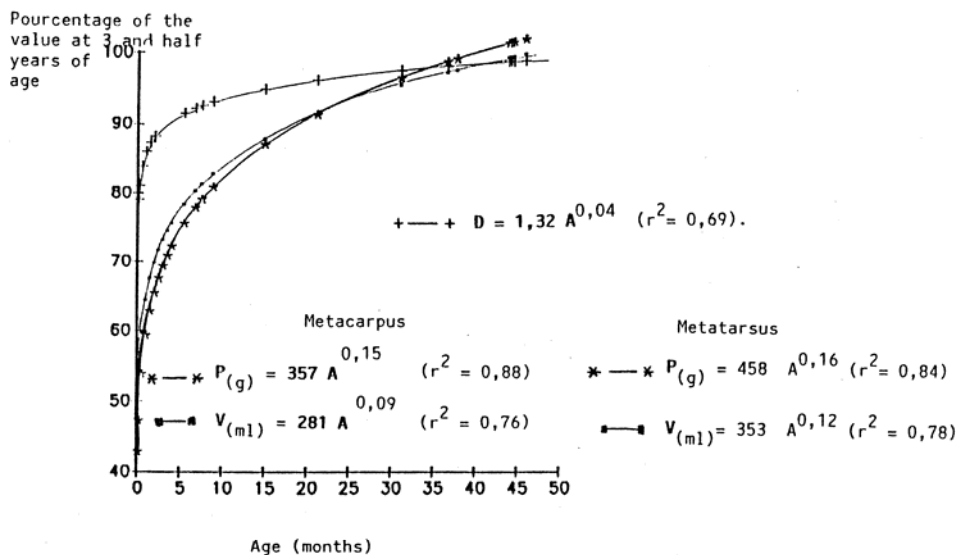


Figure 13a. Variation of physical characteristics of cannon bone with age. Increase owing to the age ( $A$ ) of weight ( $P$ ), volume ( $V$ ) and density ( $D$ ) of metacarpus and metatarsus (Bigot et al., 1990).

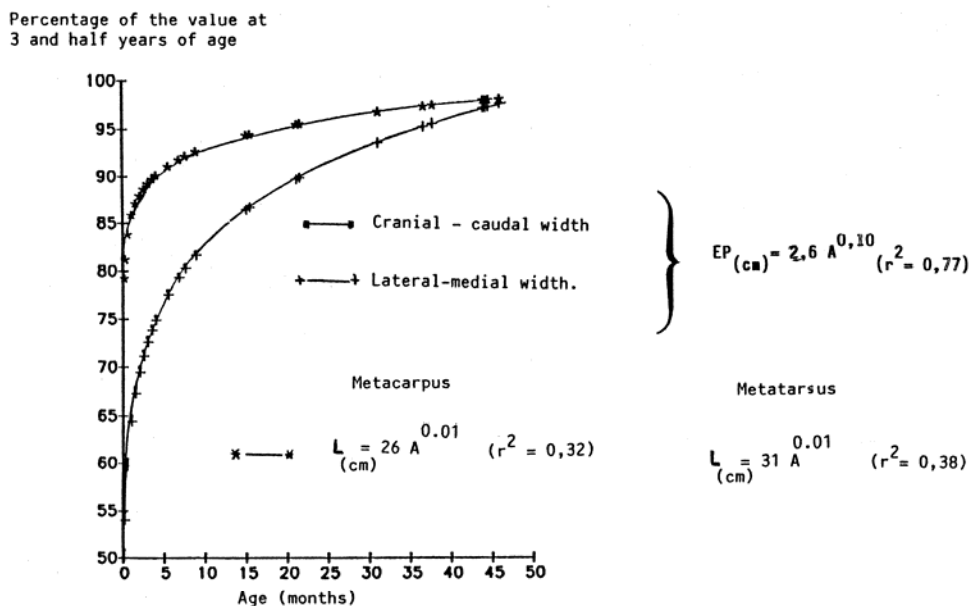


Figure 13b. Variation of physical characteristics of cannon bone with age. Increase owing to the age ( $A$ ) of length ( $L$ ), cranial-caudal and latero-medial width ( $Ep$ ) of metacarpus and metatarsus (Bigot et al., 1990).

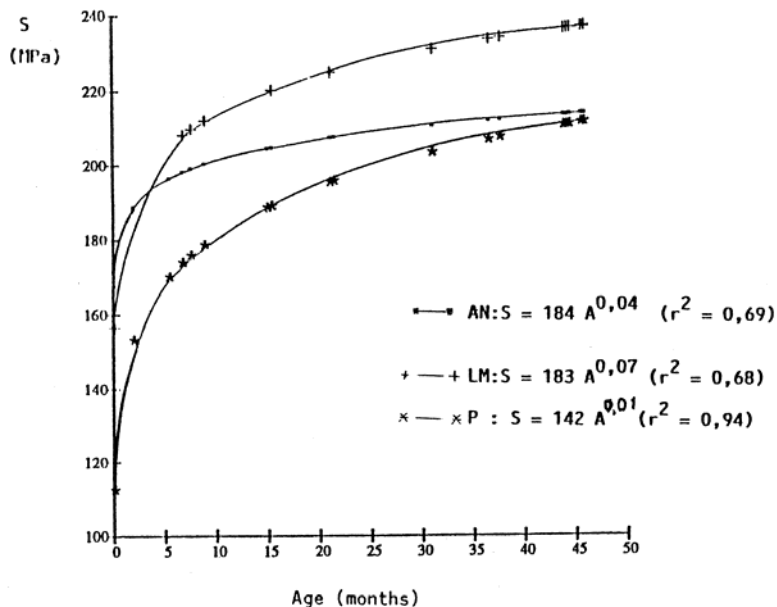


Figure 14a. Variation of mechanical properties of the cannon bone with age. Increase with age ( $A$ ) of breaking stress ( $S$ ) of cranial (AN), lateral and medial (LM), caudal (P) quadrants of cannon bone (from Bigot et al., 1990).

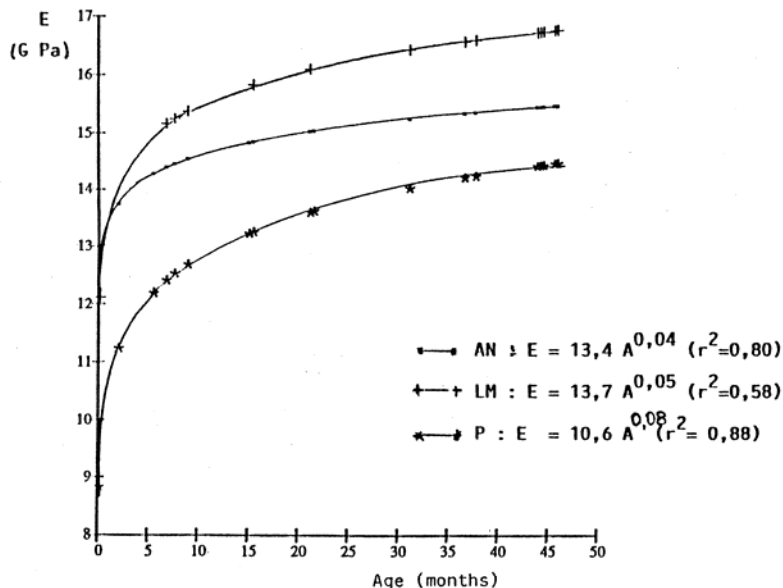


Figure 14b. Variation of mechanical properties of the cannon bone with age. Increase with age ( $A$ ) of modulus of elasticity ( $E$ ) of cranial (AN), lateral and medial (LM), caudal (P) quadrants of cannon bone (from Bigot et al., 1990).

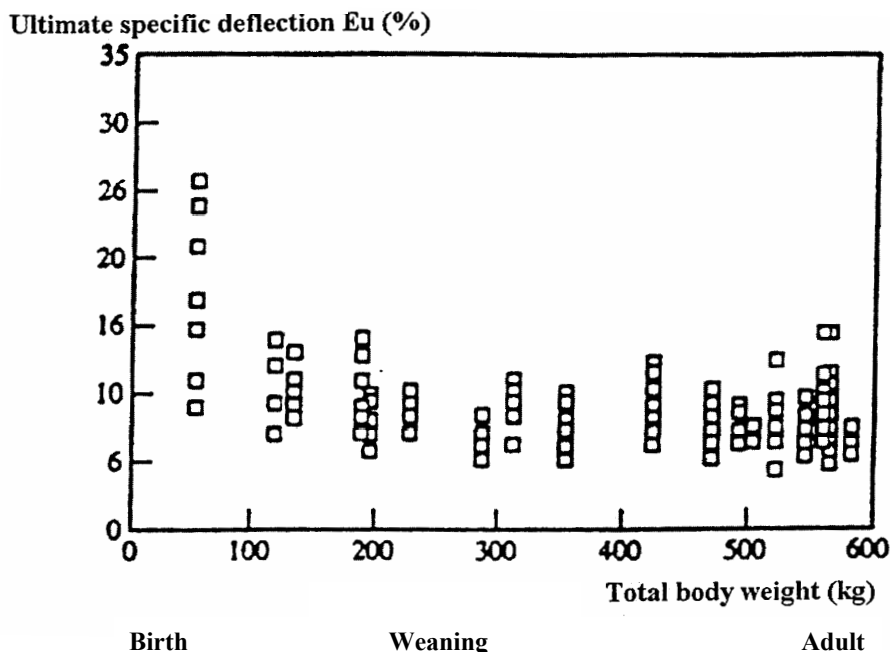


Figure 15. Evolution of ultimate specific deflection (Eu) with the total body weight of growing horses (from Bigot *et al.*, 1996).

## Factors of variation of growth and development

Growth and development are determined by genetic potential, affected by environmental factors and under the control of hormones.

### Genetic effects

In equines body size and body weight ranges between 1 and 5 as far as ponies and heavy horses are concerned.

Genetic effect on body size is high and the heritability coefficient of most body size parameters range between 0.36 and 0.49 and the average heritability coefficient for body size is  $h^2 = 0.35$  (Langlois, 1973).

Comparing heavy breeds (Marcq *et al.*, 1956) to American light breeds (Heird, 1973) it arises that heavy breeds are later maturing than light breeds (Figure 16). Arab breed would be later maturing than thoroughbred (Grabowski *et al.*, 1971). But this genetic effect has to be restricted by the maternal body size effect as the stallion *sensu stricto* effect would be 70 p.100 of maternal effect (Langlois, 1973). Such maternal effect was pointed out clearly in the experiments of reciprocal cross breeding between ponies and light or heavy breeds (Table 7).

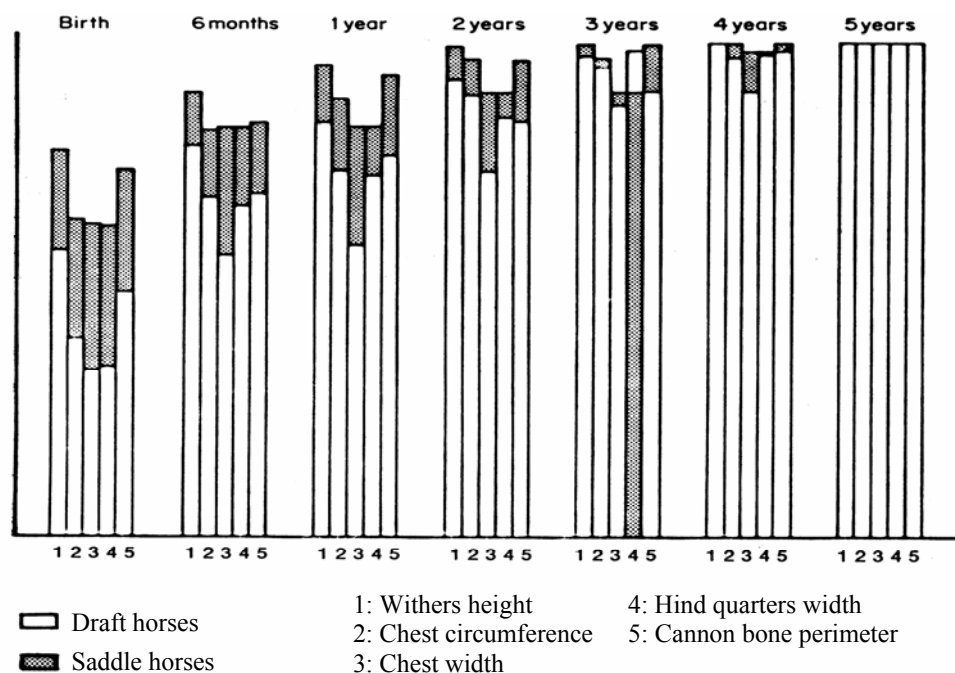


Figure 16. Comparative development of foals from draft horses (Belgian) and saddle horses (Quarter-Horse) expressed as a p. 100 of adult values (from Marcq et al., 1956; Heird, 1973).

Table 7. Cross breeding experiments in horses.

| Dam          | Sire         | Body weight at birth (kg) | References               |
|--------------|--------------|---------------------------|--------------------------|
| Shetland     | Shire        | 17                        | Walton and Hammond, 1938 |
| Shire        | Shetland     | 53                        |                          |
| Shetland     | Shetland     | 17                        |                          |
| Shire        | Shire        | 70                        |                          |
| Shetland     | Mecklenbourg | 27                        | Flade, 1965              |
| Mecklenbourg | Shetland     | 48                        |                          |
| Shetland     | Shetland     | 21                        |                          |
| Mecklenbourg | Mecklenbourg | 60                        |                          |

Genetic effect on body weight, ADG and body composition is very high. ADG is always on average 50 p.100 higher in heavy breeds than in light breeds. ADG is highly linked to adult body size of the breeds: 0.25 – 1.0 – 1.3 kg between 6 and 12 months in horses of 250 – 450 and 900 kg adult body weight (Jordan and Myers, 1972; Staun, 1973). But ADG of the five major French heavy breeds fed high feeding level range between 0.8 to 1.1kg/d only (Martin-Rosset and Jussiaux, 1977).

Comparing at the same age of slaughter, body composition of different French heavy breeds it arises that carcass weights is 10 to 17 p.100 higher in large size than in small size breeds (Robelin *et al.*, 1984). But comparing at the same empty body weight, there is no more significant difference as there is no difference in relative growth of the carcass (Table 8). The

relative growths of adipose and muscle tissues in the empty body weight are significantly higher and lower in early than late mature breeds respectively (Table 8). As a result the proportions of fat and muscles in the same carcass weight are slightly different. And the different breeds reach the same fat content in the body at different body weight which range between 471 kg and 580 kg (Martin-Rosset *et al.*, 1980). But these differences are much lower in horses than those observed in cattle (Beranger and Robelin, 1977; Robelin *et al.*, 1979).

Table 8. Variation of the relative growth and body composition in different French heavy breeds (drawn from Martin-Rosset *et al.*, 1983b).

| Animals                                                      | Breeds<br>Number<br>Mean | Ardennaise<br>13<br>Allometric coefficient | Boulonnaise<br>15  | Bretonne<br>13      | Comtoise<br>17       | Percheronne<br>15   |
|--------------------------------------------------------------|--------------------------|--------------------------------------------|--------------------|---------------------|----------------------|---------------------|
| Factorial effect on weight of                                |                          |                                            |                    |                     |                      |                     |
| Carcass                                                      | 356.1                    | 0.993                                      | 1.003              | 1.000               | 1.001                | 1.003               |
| muscles                                                      | 250.5                    | 0.985 <sup>a</sup>                         | 1.022 <sup>a</sup> | 1.006 <sup>ab</sup> | 0.970 <sup>acd</sup> | 1.019 <sup>b</sup>  |
| fatty tissues                                                | 38.4                     | 1.106 <sup>a</sup>                         | 0.794 <sup>b</sup> | 0.998 <sup>ab</sup> | 1.320 <sup>ac</sup>  | 0.864 <sup>ab</sup> |
| bones                                                        | 54.6                     | 0.968 <sup>a</sup>                         | 1.064 <sup>b</sup> | 1.004 <sup>ad</sup> | 0.915 <sup>bc</sup>  | 1.057 <sup>bd</sup> |
| Carcass composition for 356 kg carcass weight                |                          |                                            |                    |                     |                      |                     |
| muscles                                                      |                          | 69.2                                       | 71.9               | 70.8                | 68.2                 | 71.7                |
| carcass fatty tissues                                        |                          | 11.9                                       | 8.6                | 10.8                | 14.2                 | 9.3                 |
| bones                                                        |                          | 14.8                                       | 16.3               | 15.3                | 14.0                 | 16.2                |
| Percent composition of total muscles at 250 kg muscle weight |                          |                                            |                    |                     |                      |                     |
| neck                                                         |                          | 11.5                                       | 11.7               | 11.8                | 11.9                 | 11.7                |
| thorax                                                       |                          | 17.7                                       | 17.9               | 18.0                | 18.1                 | 17.3                |
| spinal column                                                |                          | 12.0                                       | 12.1               | 11.8                | 11.9                 | 12.4                |
| column                                                       |                          | 3.3                                        | 3.5                | 3.3                 | 3.3                  | 3.3                 |
| abdominal wall                                               |                          | 6.5                                        | 6.3                | 6.3                 | 6.7                  | 6.3                 |
| fore-limb                                                    |                          | 13.7                                       | 13.5               | 13.5                | 13.4                 | 13.4                |
| hind-limb                                                    |                          | 38.2                                       | 38.2               | 38.3                | 37.6                 | 38.5                |

a,b,c,.....x<sup>a</sup>, y<sup>b</sup>: values with unlink subscripts are significantly different.

The variation in relative growth of muscles is less significant between light breeds (Table 9).

Table 9. Relative growth of main muscle regions as compared to live weight in saddle horses and thoroughbred (from Gunn, 1975).

| Muscle regions | Allometric coefficient |              |
|----------------|------------------------|--------------|
|                | Saddle horse           | Thoroughbred |
| Fore limb      |                        |              |
| Distal         | 1.04                   | 1.02         |
| Proximal       | 1.01                   | 1.05         |
| Chest          | 0.99                   | 1.04         |
| Hind limb      |                        |              |
| Distal         | 0.97                   | 1.11         |
| Proximal       | 1.05                   | 1.15         |

Y = Muscle regions

X = Body weight

## Hormonal factors

### Growth hormone (GH)

The growth hormone is produced in the anterior part of the hypophyse. GH is known in the mammals to increase the growth rate, the bone growth via the metaphyseal growth plate, the lipolysis of adipose tissues and the protein anabolism.

The length of long bone is increased by GH as far as the differentiation and the proliferation of chondrocytes on the growth plate are stimulated directly (Isaksson and al., 1982; Isgaard *et al.*, 1986; Lindhal *et al.*, 1986) and via IGF-1 (Isaksson *et al.*, 1987) respectively. GH stimulates collagen and interstitial protein synthesis and supply of energy from fatty acids to support the multiplication of chondrocytes too. IGF-1 enhances collagen synthesis by osteoblasts (Schmid *et al.*, 1983).

The role of GH and its interactions with other hormones have been evidenced by work conducted at INRA in the nineties. In the foal, GH secretion is pulsatile but it is not linked to the suck (Benedit *et al.*, 1990) conversely to what is observed in the suckling calve (Coxam *et al.*, 1987). During the suckling period, GH and IGF-1 secretion is high and associated to high concentration of osteocalcin of plasmatic calcium and phosphorus (Figures 12, 17 and 18). An injection of GRF in the foal one week after birth induces an increase in GH concentration, which evidence the stimulation of the GH secretion by GRF (Davicco *et al.*, 1993).

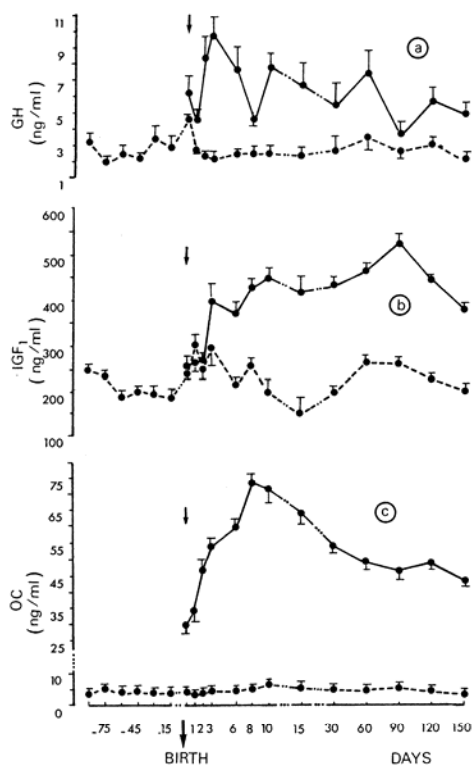


Figure 17. Plasma GH (a), IGF1 (b) and osteocalcin (c) concentration in foals (—) and mares (---) (Davicco *et al.*, 1994).

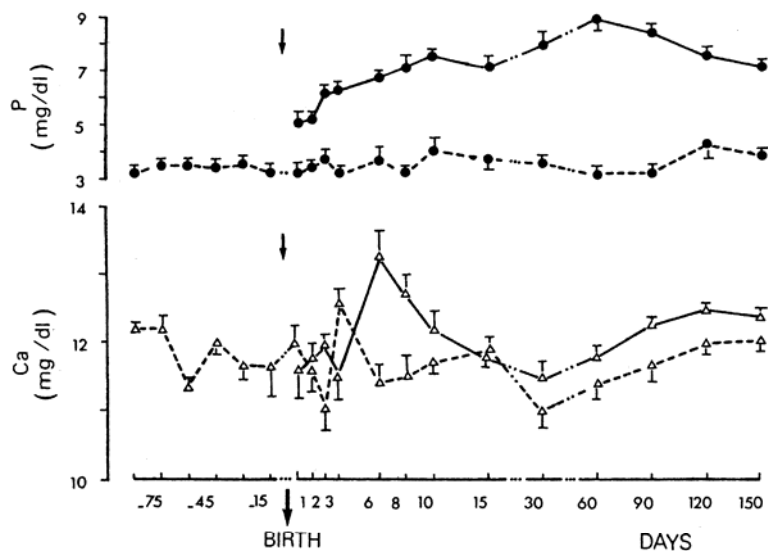


Figure 18. Plasma calcium ( $\Delta$ ) and inorganic phosphorus ( $\bullet$ ) concentration in foals (—) and mares (---) (Davicco et al., 1994).

### Thyroxines

In human, lack of thyroxines induces strong physiological and metabolic disorders: cretinism, reduction in basal metabolism, break of growth and sexual development...

Direct and indirect effects of GH are stimulated by thyroid hormones ( $T_3$  and  $T_4$ ). The triiodothyronine ( $T_3$ ) promote the differentiation of chondrocytes, the maturation of growth plate (direct effect of GH) and the proliferation of chondrocytes (indirect effect of GH – Burch and Van Wyk, 1987). The thyroxine ( $T_4$ ) would stimulate the longitudinal growth of bone as far as GH effect would promote this influence (Thorngren and Hanson, 1973c).

### Sexual hormones

The action and the influence of the sexual hormones (androgens, oestrogens and progestagens) rise with the sexual development in all the animals. The influence of androgens is direct whereas the influence of estrogens is via the hypophyse. The androgens stimulate the protein synthesis and the progestagens increase the growth (gravid anabolism).

Female would be early maturing than males either in heavy or light breeds (Marcq *et al.*, 1956; Heird, 1973). But the differences in body size would depend on the region (Figure 19). At on year of age chest width and height of hind quarters, hind quarter length and trunk length are higher in female than in males. Conversely the development of forelimbs is earlier in males than in females (Heird, 1973). The cannon perimeter would be higher in males than in females at 18 months in polish half-warm blood (Brzeski and Kurowski, 1964) and in Arab (Grabowski and al, 1971) and only at 2.5 years old in Belgian draft breed (Marcq and al, 1956). Recent data would point out that genetic effect would be significant at 20 months of age in light breeds (Paragon *et al.*, 2000). Adult body size of males is 10 p.100 higher than in female (Figure 19 and Butaye, 1966). Body weight evolution is not significantly different



until 18 or 30 months in stallions or gelding of heavy breeds respectively (Olsson, 1952; Martin-Rosset and Jussiaux, 1977) or 24 months in light breeds (Grabowski *et al.*, 1971; Paragon *et al.*, 2000). At adult age the stallions are on average 10 p.100 heavier than the mares. But most of the males are castrated between 12 and 24 months. In the heavy breeds, the effect of a castration at 12 months occurs only at 30 months but the difference in body weight is only 5 p.100 (Yablakov, 1976).

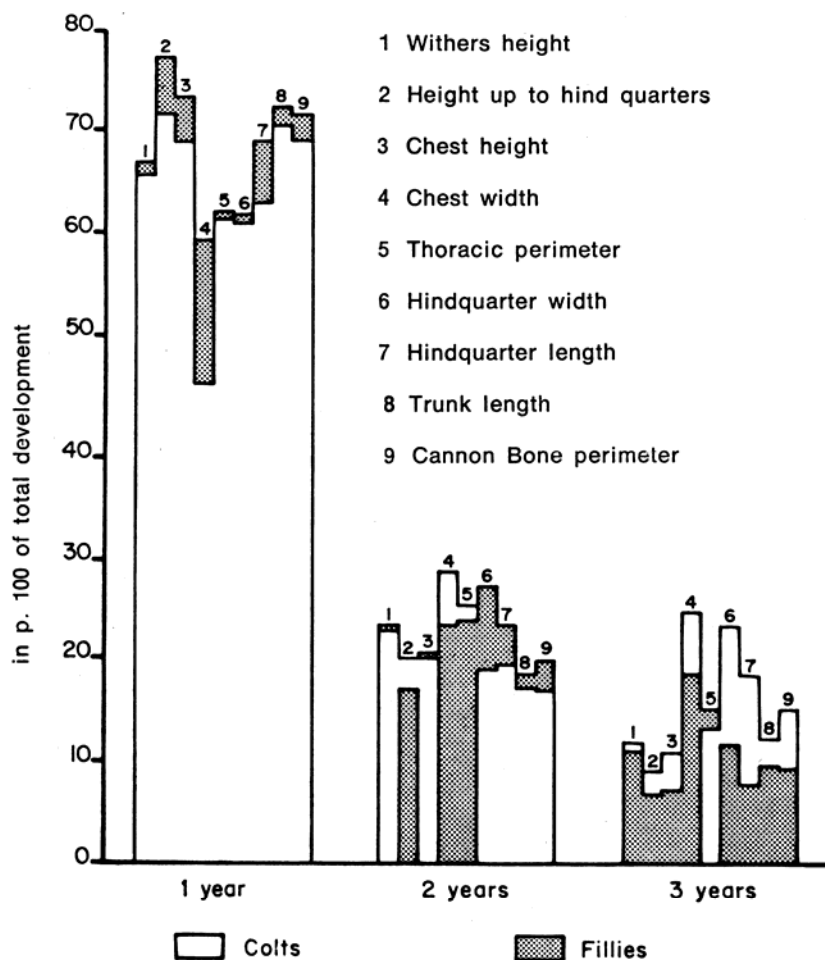


Figure 19. Effect of sex on development of various body areas in Belgian draft horse (from Marcq *et al.*, 1956).

In heavy breeds, empty body weight and carcass weight, are both on average 10 p.100 higher in males than in females between 12 and 30 months of age (Robelin *et al.*, 1984). The proportion of adipose and muscle tissues in the carcass are higher (+ 30 p.100) and slightly lower (-1 p.100) respectively in females than in males but the difference is not significant; where as the relative growth of muscle is slightly higher ( $b=+0.15$ ) in the females the males. The relative growth of adipose tissues is not significantly different and the distribution of adipose or muscle tissues is similar in both sexes (Table 10).

Table 10. Influence of sex on body composition and relative growth of various muscular regions. (drawn from Martin-Rosset *et al.*, 1983b).

| Animals                                                               | b male<br>n=39    | b female<br>n=34  |
|-----------------------------------------------------------------------|-------------------|-------------------|
| Allometric coefficients                                               |                   |                   |
| Carcass                                                               | 1.04              | 1.04              |
| Muscles                                                               | 0.91 <sup>a</sup> | 1.04 <sup>b</sup> |
| Fatty tissues                                                         | 2.13              | 2.13              |
| Bones                                                                 | 0.71              | 0.71              |
| Body composition at 356 kg carcass weight                             |                   |                   |
| Muscles                                                               | 70.7              | 70.0              |
| Carcass fatty tissues                                                 | 9.4               | 12.3              |
| Bones                                                                 | 15.7              | 14.9              |
| Compared percentage of muscle weight at 250 kg to total muscle weight |                   |                   |
| Neck                                                                  | 12.1              | 11.4              |
| Thorax                                                                | 17.8              | 17.8              |
| Spinal column                                                         | 12.2              | 12.0              |
| Abdominal wall                                                        | 6.3               | 6.5               |
| Fore-limb                                                             | 13.5              | 13.5              |
| Hind-limb                                                             | 37.9              | 38.4              |

b: allometric coefficient

a, b, .....x<sup>a</sup>, y<sup>b</sup> – variables with unlike subscripts are significantly different

Longitudinal growth and bone maturation are enhanced by sexual hormones whereas the castration delays them and induces an osteopeny (Morsher, 1968). At the puberty a heavy endogen secretion of sexual hormones closes the growth, which induces the closing of the growth plates. In the rat, the multiplication of chondrocytes is closed by estrogens at the level of growth plate (Gustavsson *et al.*, 1975) but the maturation of chondrocytes and the calcium deposition in the cartilaginous matrix are enhanced (Hoshino and Olsson, 1975). In the new born and one month old foal, GH secretion is stimulated par the intravenous injection of sexual steroids: testosterone or 17  $\beta$ -oestradiol whereas it is reduced by the injection of corticoids (Cortisol) (Davicco *et al.*, 1992).

## Effect of nutrition

### Influence of the feeding level during the winter periods

The body weight of light breeds rises with the increase in the feeding level (Figure 20; Table 11a). This relationship is implemented in the nutritional models, which have been performed by INRA to evaluate energy and protein requirements (Martin-Rosset *et al.*, 1994):

$$\begin{aligned}\text{Energy} &= a\text{BW}^{75} + b\text{BW}^{75}\text{G}^{1.6} \\ \text{Protein} &= a\text{BW}^{75} + b\text{G}\end{aligned}$$

Similar figures were observed in heavy breeds (Martin-Rosset *et al.*, 1994). But the influence of the feeding level is decreasing as much as the growing horse is getting older and whatever the breeds (Figure 20). The influence of the feeding level on the body size is less effective mainly in the late age (Table 11b).

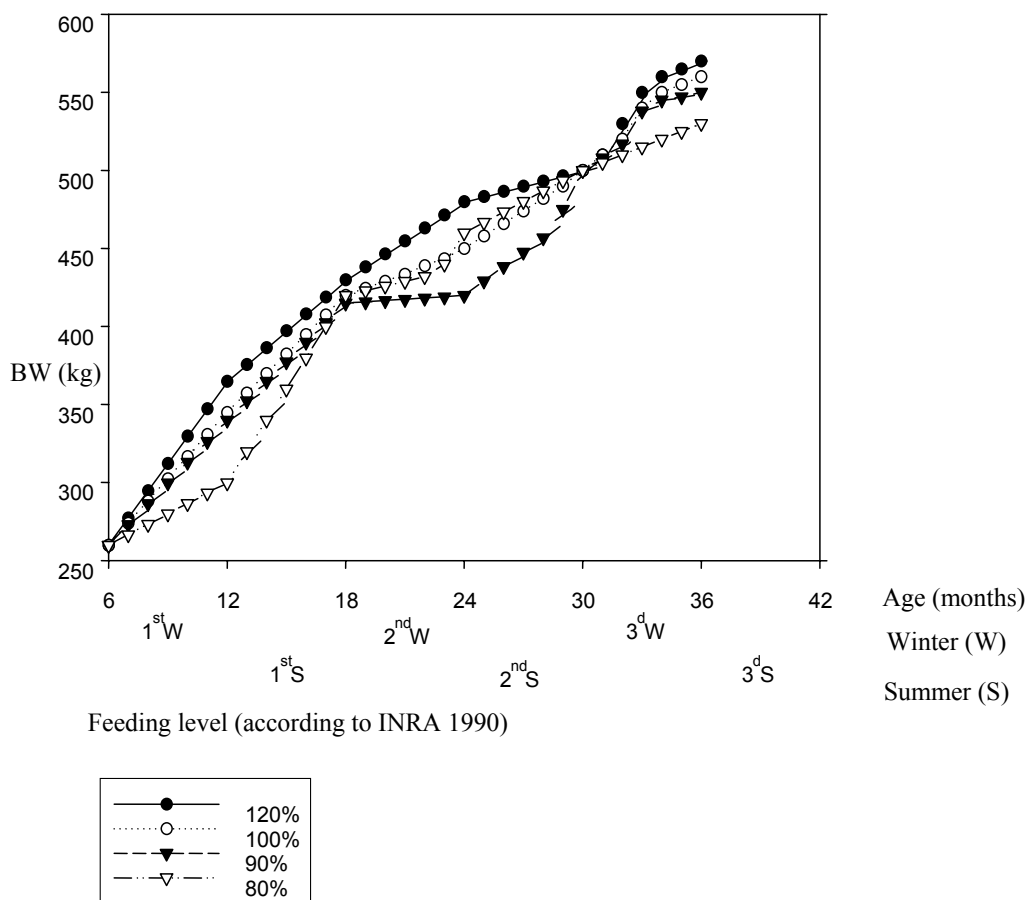


Figure 20. Influence of the level of feeding on body weight during winter periods (drawn from Bigot et al., 1987).

### Modulation of the feeding level of growing horse fed indoor

Body weight and size of young horses can be strongly affected by a very wide difference in the feeding level: potential versus linear models namely if the foals are light at weaning as pointed out in feeding experiments carried out at INRA between 6 and 36 months (Figure 21a). 4 groups of animals heavy or light at weaning were conducted according a curvilinear or linear model of growth. The variation in body size is similar to that of body weight. The heavy animals at weaning conducted according to potential or linear models reach the same body weight and size at 36 months. The light animals at weaning reach nearly the same body weight at 36 months. But the body size of the latest is always lower than the heavy ones at weaning. The body size of the light animals at weaning conducted according to a potential model is always lower than the sizes of the other groups mainly after 2 years of age where the body size plateau. (Figure 21b).

Table 11. Influence of feeding level on growth and development in horses of light breeds (Anglo Arab – Selle Français) (drawn from Bigot et al., 1987).

Table 11a. Average daily gain during 3 subsequent winter-feeding period between weaning and 42 months: effect of feeding level.

| Feeding level  | Low level            |                  |                      |                  |                      |                  | High level           |                  |                      |                  |                      |                  |
|----------------|----------------------|------------------|----------------------|------------------|----------------------|------------------|----------------------|------------------|----------------------|------------------|----------------------|------------------|
|                | 1 <sup>st</sup> year |                  | 2 <sup>nd</sup> year |                  | 3 <sup>rd</sup> year |                  | 1 <sup>st</sup> year |                  | 2 <sup>nd</sup> year |                  | 3 <sup>rd</sup> year |                  |
|                | Winter               | Summer           | Winter               | Summer           | Winter               | Summer           | Winter               | Summer           | Winter               | Summer           | Winter               | Summer           |
| Years of birth |                      |                  |                      |                  |                      |                  |                      |                  |                      |                  |                      |                  |
| 1979           | 589 <sup>a</sup>     | 690 <sup>a</sup> | 259 <sup>a</sup>     | 344 <sup>c</sup> | 71 <sup>a</sup>      | 278 <sup>c</sup> | 389 <sup>b</sup>     | 837 <sup>b</sup> | 1 <sup>b</sup>       | 514 <sup>c</sup> | -43 <sup>b</sup>     | 315 <sup>c</sup> |
| 1980           | 528 <sup>a</sup>     | 159 <sup>a</sup> | 365 <sup>a</sup>     | 529 <sup>c</sup> | 18 <sup>a</sup>      | 365 <sup>c</sup> | 231 <sup>b</sup>     | 658 <sup>a</sup> | 130 <sup>b</sup>     | 680 <sup>c</sup> | 0 <sup>a</sup>       | 419 <sup>c</sup> |
| 1981           | 751 <sup>a</sup>     | 537 <sup>a</sup> | 749 <sup>a</sup>     | 36 <sup>c</sup>  | 370 <sup>a</sup>     | 5 <sup>c</sup>   | 535 <sup>b</sup>     | 544 <sup>a</sup> | 547 <sup>b</sup>     | 156 <sup>d</sup> | -24 <sup>b</sup>     | 40 <sup>c</sup>  |
| 1982           | 942 <sup>a</sup>     | 525 <sup>a</sup> | 405 <sup>a</sup>     | 3 <sup>c</sup>   | 522 <sup>a</sup>     | 144 <sup>c</sup> | 820 <sup>a</sup>     | 607 <sup>a</sup> | 67 <sup>b</sup>      | 319 <sup>d</sup> | 336 <sup>d</sup>     | 299 <sup>f</sup> |

For a same period ADG affected by different superscripts are statistical different.

Table 11b. Variation of body size (cm) during 3 subsequent winter feeding periods (between weaning and 42 months in horses of light breeds). Effect of feeding level (drawn from Bigot et al., 1987).

| Basal diet   | Year of birth | Age (d) | FL   | 1 <sup>st</sup> winter |         |         |         | 2 <sup>nd</sup> winter |         |         |         | 3 <sup>rd</sup> winter |         |         |         |
|--------------|---------------|---------|------|------------------------|---------|---------|---------|------------------------|---------|---------|---------|------------------------|---------|---------|---------|
|              |               |         |      | Duration (d)           | HW (cm) | TH (cm) | TL (cm) | Duration (d)           | HW (cm) | TH (cm) | TL (cm) | Duration (d)           | HW (cm) | TH (cm) | TL (cm) |
| Hay + Straw  | 1979          | 269     | Low  | 94                     | 6.70    | 3.50    | 2.15    | 140                    | 3.70    | 0.60    | -0.10   | 118                    | 1.00    | 1.00    | -0.40   |
|              |               |         | High |                        | 5.15    | 2.30    | 1.45    |                        | 2.45    | 0.85    | 0.85    |                        | 0.45    | 1.00    | -1.05   |
|              | 1980          | 250     | Low  | 122                    | 5.30    | 3.10    | 0.20    | 101                    | 0.45    | 0.75    | 0.25    | 118                    | 1.50    | 0.85    | -0.65   |
|              |               |         | High |                        | 5.80    | 3.61    | 1.22    |                        | 2.17    | 1.67*   | 1.33    |                        | 1.89    | 0.66    | -0.66   |
| Maize silage | 1981          | 240     | Low  | 129                    | 8.96    | 5.65    | 2.87    | 118                    | 2.37    | 2.75    | 3.54    | 137                    | 0.79    | 1.33    | 1.98    |
|              |               |         | High |                        | 7.45    | 3.60**  | 2.75    |                        | 3.35    | 2.95    | 5.20*   |                        | 0.15    | 1.45    | 2.85*   |
|              | 1982          | 241     | Low  | 118                    | 9.89    | 6.67    | 5.61    | 96                     | 0.50    | 0.61    | 0.53    | 126                    | 2.83    | 4.00    | 3.46    |
|              |               |         | High |                        | 9.22    | 6.14    | 5.07    |                        | 1.28    | 1.79    | 1.00    |                        | 1.14**  | 1.36    | 3.50    |

Statistical difference between feeding level each year of birth \* P < 0.05; \*\* P < 0.02

HW = Height at withers; TH = Thoracic Height; TL = Thoracic Length

In conclusion, the growing horse is capable to compensate a moderate delay in body weight and size after weaning, as far as the feeding level is increased according to the level and the duration of feeding restriction, during the previous period, and if the body weight at weaning is not too limited (Bigot *et al.*, 1987; Trillaud-Geyl *et al.*, 1992). The variation in body size between 6 and 36 months is directly linked to body weight variation. Age and body weight at weaning seems to be of major concern. Growing horse fed a moderate and constant feeding level are capable to meet the same body weight and size at the same age. Those recent data are quite consistent with previous data (Marcq *et al.*, 1956; Witt and Lhose, 1965; Schwark, 1968)

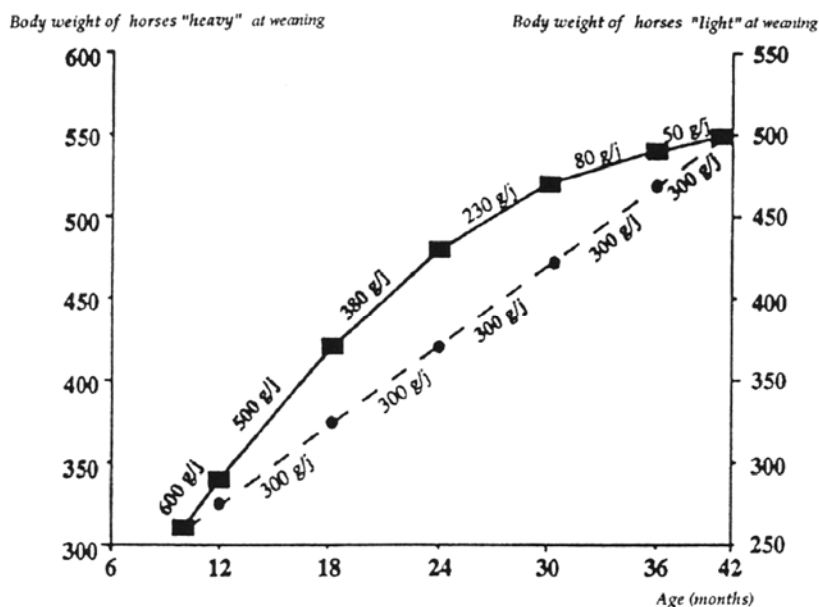


Figure 21a. Growth curve for group "potential growth" (—) and group "linear growth" (--) (from Trillaud-Geyl et al., 1992).

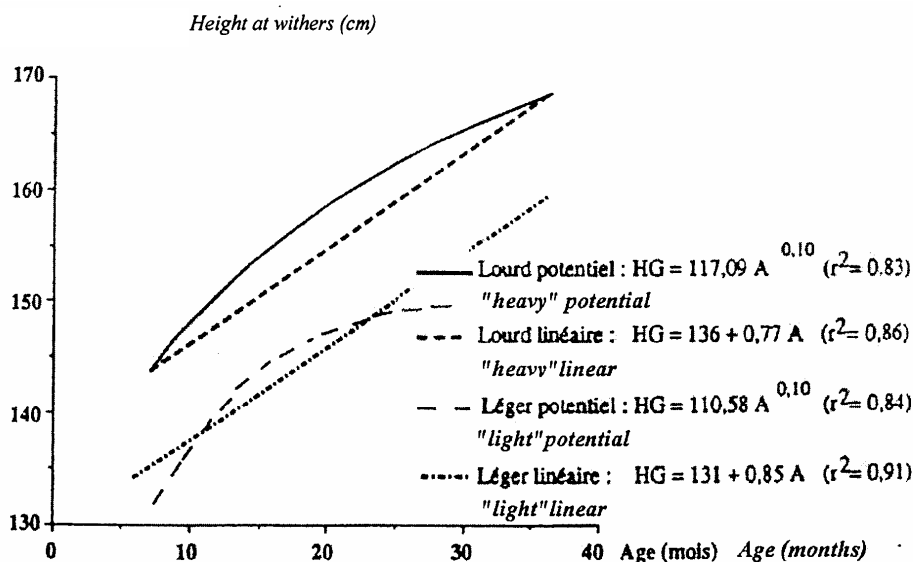


Figure 21b. Height at withers increase with age in the 4 groups in young horse (from Trillaud-Geyl et al., 1992).

## Compensatory growth

The growing horses fed moderate versus high level during the three subsequent winters between 6 to 42 months are capable to meet the same body weight and size as far as they are grazing good quality and *ad libitum* grass (Table 11). In summer, ADG of restricted animals are much higher than those of well-fed animals. But the compensatory growth during the summer is linked to the feeding level (e.g. ADG in winter) and the duration of restriction in the previous winter according to the following equation (Martin-Rosset *et al.*, 1984):

$$y = 93.39 + 6.45 x_1 \times x_2$$

y = average daily gain at grazing (kg/d)

x<sub>1</sub> = average daily gain in winter (kg/d)

x<sub>2</sub> = duration of the period of restriction (days)

And the capacity of compensatory growth decreases with age (Table 11-Figure 20).

## Influence of feeding level on tissue development

### *Bone tissue and mechanical characteristics*

The influence of 2 linear models of growth (0.450 kg: group A versus 0.350 kg: group B) were studied at INRA between 6 and 24 months of age (Table 12). At 24 months BW was 10 p.100 higher in group A than in group B and height at withers and the thickness of cannon bone were not significantly different. But the thickness of the cortex and of the inertia moment were 10 and 37 p.100 higher respectively in group A than in group B. As a result bone tissue and its mechanical characteristics are affected by a too limited body growth even though body size limitation is not associated.

*Table 12. Effect of 2 levels of growth on bone development in young horse of light breeds from 8 to 24 months of age (drawn from Bigot and Martin-Rosset, unpublished).*

|                                                          | Group A | Group B | Difference (B/A) % |
|----------------------------------------------------------|---------|---------|--------------------|
| Daily gain (g/d)                                         | 350     | 450     |                    |
| Body weight (kg):                                        |         |         |                    |
| 8 months (kg)                                            | 301     | 300     | -                  |
| 24 months                                                | 471     | 520     | +10                |
| Body size:                                               |         |         |                    |
| Height at withers (cm)                                   | 159.1   | 159.8   | -                  |
| Width at shoulders (cm)                                  | 41.4    | 45.4    | +10                |
| Physical and biochemical characteristics of cannon-bone: |         |         |                    |
| Bone thickness (cm)                                      | 4.43    | 4.51    | -                  |
| Cortex thickness (medial area)                           | 11.7    | 14.1    | +20                |
| Inertia moment (cm <sup>4</sup> )                        | 12.9    | 17.7    | +37                |

## Adipose tissues

The proportion of total adipose tissues in the carcass of colts of heavy breeds slaughtered at 12 months is 22p100 (e.g. +2.2 points) higher in the group fed 30p100 more energy than in control group whereas the proportion in muscles is 4p100 lower (e.g. -2.5 points) (Martin-Rosset and Jussiaux, 1977; Robelin *et al.*, 1984).

## Effect of the type of diet

### Body weight

In suckling horses of light or heavy breeds body weight is depending of milk intake as ADG is linked to milk yield according to the following equation (Doreau *et al.*, 1986) until 2 months of age:

$$\text{ADG (kg/d)} = a \times X$$

a= kg of milk/kg ADG

a=10.6 at one month of age R=0.81

a=13.7 at two months of age R=0.74

X= ADG (g/d)

Then ADG decreases as far as the foal is increasingly grazing.

In weanling horses of light and heavy breeds, ADG is as much high as the foal is supplemented + 18p100 when the foal are fed 2kg concentrates since 4 months of age (Martin-Rosset and Trillaud-Geyl, 1984), and according the type of supplement (Table 13) and that limiting amino acids requirements are met (Hintz *et al.*, 1971; Breuer and Golden, 1971; Pulse *et al.*, 1973; Yoakam *et al.*, 1978; Ott *et al.*, 1979). Comparing foals weaned at 6 or 4.5 months of age, ADG and BW at 6 months of age would not be significantly different (Warren *et al.*, 1998). But ADG of foals born in spring would be higher than ADG of foals born in winter due season effect: e.g. the influence of the higher nutritive value and intake of grass in spring than hay in winter (Pagan, 1998).

Table 13. Influence of the type of supplementation on the growth of weanling of light breeds (drawn from Borton *et al.*, 1973).

| Diet (7 weeks)          | Soya              | Skimmed milk       | Soya              | Skimmed milk      |
|-------------------------|-------------------|--------------------|-------------------|-------------------|
| Crude protein content)  | (14 %)            | (14 %)             | (22 %)            | (14 %)            |
| Nr animals              | 6                 | 6                  | 6                 | 6                 |
| Average daily gain (kg) | 0.60 <sup>c</sup> | 0.76 <sup>a</sup>  | 0.65 <sup>c</sup> | 0.95 <sup>a</sup> |
| Growth height (cm)      | 5.08 <sup>c</sup> | 6.60 <sup>cc</sup> | 6.99 <sup>d</sup> | 7.19 <sup>d</sup> |

<sup>a,b,c,d</sup> Values with unlike superscript are significantly different ( $P < 0.01$ )

### Body weight and body composition

Foals of heavy breeds fed *ad libitum* a hay or maize silage diet supplemented with the same amount of concentrate are 5 p.100 heavier where fed maize silage and fat content in the carcass is 22 p.100 higher too (Robelin *et al.*, 1984).

## Effect of exercise

Bone tissue synthesis is induced by moderate exercise whereas there is a remodelling by osteoclasts as far as exercise is reduced (Dalin and Jeffcott, 1994). The process is reversible. There would be a curve for response of intensity effect and threshold for optimal efficiency in horse as in human. But it has to be determined. The thickness of the cortex in the caudal quadrant of the cannon bone and the bone density increase with exercise (Nunamaker *et al.*, 1989; Mc Carthy *et al.*, 1991-1992). The collagen fibres are oriented towards the same lines in the exercising horse (Riggs and Evans, 1992; Riggs *et al.*, 1993) whereas linkage between the collagen fibres would rise and contribute to the regulation of mineralization. As a result mechanical properties of the cannon bone is improved by exercise until an optimum, which has to be determined.

## Discussion and conclusion

### Variation of Body Weight (BW) and Body Size (BS)

From the fertilization to adult age, BW and BS increase with age in horse as in other mammals. During the pregnancy BW and BS increase exponentially. Average daily gain (ADG) of the foetus in horse 500kg mature is 1500g/d. Comparing to other mammals, BW at birth is higher relatively to adult BW in horse than in cattle: 9-10p100 versus 6-8p100 respectively. ADG during the first postnatal year is high 2000-800g/d, mainly during the very early period, and total BW gain accounts for 50-60p100 of adult BW. Then ADG decrease in horse as in cattle but faster. Similarly the increase in BS is very high: height at withers rise by 70p100 during the first postnatal year. Comparing with other farm animals the horse seems to be an early animal species.

ADG of light breeds is lower than in heavy breeds. But differences between the light breeds groups are not known whereas the differences between the heavy breeds group seem to be more limited than in cattle: 100-200g/d and 200-500g/d respectively. BW and BS are higher in males than in females. But all these figures should have to be refined in experiments especially designed for that purpose and until the adult BW and BS to determine accurately age at maturity and precocity at the point of inflection, using the appropriate model. Such work is in progress in France at INRA and Haras Nationaux using data obtained from birth until 4 years of age in sports horses conducted in long feeding experiments. BW and BS can be strongly affected by feeding level. BS is strongly influenced by the variation in BW due to the level of feeding and the duration of the period of restriction or/and probably of overfeeding, as much as the age where restriction or overfeeding take place.

### Tissue development

After birth the development of skeleton and most of the organs, except the stomach and the large intestine, is very early whereas that of adipose tissues is late but increasingly high with age. The development of muscle is very close to that of carcass in empty body weight. All these figures are consistent with the general pattern defined by Hammond. The proportion of skeleton in empty body weight decreases with age whereas the proportion of muscles and namely adipose tissue increase as in cattle. The relative growth of skeleton or muscles is earlier in distal limbs and later in proximal limbs and trunk respectively. After birth, lipids content increases whereas the water content decreases. But the composition of free fat body mass seems to be nearly stable.



Breeds and sex influence body composition. The relative difference in the proportion of adipose tissue in the carcass can range 19 to 65p100 as far as the different breeds are compared at the same carcass weight. But the variation in muscle proportion is very low, no more than 5p100. And there is no great variation in the proportion of the different muscle regions. The adipose tissue proportion can be 30p100 higher in female than in male as far as the animals are compared at the same carcass weight. But there is no significant variation in total muscles and of muscles regions. The type and level of feeding significantly affect the proportion of adipose tissue (15 to 30p100). as it is strongly linked with ADG .

All these figures have been described in heavy breeds slaughtered at different ages after birth. We can expect that the general pattern of tissue which have been described can be applied to light breeds as far as the pattern is very consistent with that of Hammond.

Adipose tissue is the most variable tissue in horse as in cattle, with age, breed, sex and nutrients intake. Lipids are costly components to be synthesised. As a result lipids content is of high concern in the determination of nutrients requirements of growing horse.

Bone tissue development develops very early in horse. As a result bone tissue is strongly influenced by nutrients intake e.g. feeding management of the mare and of the foal. Age, BW and BS are of major concern at weaning for the further bone growth and characteristics. Bone tissue structure and mechanical properties are influenced by exercise but the curve of response has to be designed in horses.

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# Physiology of growth and development

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## Abstract

Musculoskeletal disorders rank first among the ailments that may lead to untimely retreat from athletic activity in horses. Of these, disorders concerning articular cartilage and tendons have most impact because of their high incidence and their notoriously bad tendency to heal. This bad healing tendency is related to the low metabolic rate and, consequently, high turnover times of components of the extracellular matrix (ECM), predominantly collagen, in mature animals. Recent research has shown that in mature animals there is a close match between biochemical composition of the ECM and biomechanical challenging during locomotion and athletic activity, the so-called “functional adaptation” of the tissue. In the new-born animal such an adaptation does not yet exist, but is formed in the early post-natal period under the influence of articulation and weight-bearing. As the collagen network will not be able to change or remodel substantially after a given age due to the rapid decline in metabolism, this early period during which the ECM is shaped, is of crucial importance for tissue quality and hence for resistance to injury during the entire life of the animal. Conditions in the early post-natal period (nutrition and exercise) are thus much more important for the prevention of musculoskeletal disorders than previously thought.

*Keywords: foal, development, functional adaptation, cartilage extracellular matrix, musculoskeletal disease, exercise, osteochondrosis*

## Introduction

The horse has been domesticated about 5,000 years ago, probably anywhere in what is now Southern Russia (Dunlop and Williams 1996). Unlike many other species that had been domesticated before, such as small and large ruminants, pigs and poultry, the horse was not domesticated for a tangible product such as meat, milk, hides or wool that could be either eaten or used for clothing or for the preparation of other tools. The horse’s “product” was its unequalled locomotion capacity that would play an important role in the history of mankind during the next five millennia. It can be stated that the introduction of the horse was the greatest revolution in warfare before the invention of gunpowder in the 14<sup>th</sup> century AD (van Weeren, 2000). However, the horse became an indispensable part of society for more peaceful purposes as well: in agriculture and civil transport the horse has been pivotal until very recently. After the process of mechanisation, that started during the First World War and that in most of the world ended shortly after World War II, the horse had lost all three of its classical roles in society. With the numbers of horses dwindling, some comments were made in the 1950s that the glory period of the horse was over (Grogan, 1951), but we now know that the species has made an extremely successful comeback as a sports and leisure animal. In its new function, as so-called *equine athlete*, it is again the locomotion system that determines the value of the horse for human society.

Given the fact that its locomotion system can be considered the *raison d'être* of the horse, it is no surprise that ailments of this system rank first among the disorders that affect the species and that may severely impair the usefulness of individual animals, leading to untimely retreat from athletic activity and, consequently, in many cases also to untimely death. Therefore, disorders of the locomotion system can be considered as major problems, both economically and in terms of animal welfare. Of the disorders of the equine musculoskeletal system, those affecting articular cartilage and tendons and ligaments are by far most important. These are not only the tissues that are most frequently affected (Rosedale *et al.*, 1985, Todhunter 1992, Williams *et al.*, 2001), but these are also the tissues that, in the mature animals, are known to hardly, if ever, regain their original quality and resistance (Watkins, 1999). Both tissue types are characterised by a large extracellular matrix (ECM) to cell ratio, a sparse or even absent (cartilage) vascularisation, and, probably related to these factors, a very long regeneration time. The fact that articular cartilage is very fractious to repair was already noted in the middle of the 18<sup>th</sup> century by William Hunter, who stated that: “*an ulcerated cartilage is universally allowed to be a very troublesome disease; that it admits of a cure with more difficulty than a carious bone; and that, when destroyed, it is never recovered*” (Hunter, 1743).

Recent research on functional biochemistry and biomechanics of equine articular cartilage and tendons, and on the developmental characteristics of these tissues, has led to a new concept of how these tissues are formed and conditioned in the young growing animal and what the consequences may be for the mature individual in terms of injury resistance and susceptibility to degenerative diseases later in life. In this paper the focus is on articular cartilage. After a short overview of the most important aspects of articular cartilage structure and function, details are given of recent research in the area. The paper ends with a discussion of the novel concept of the dynamics of this tissue and, more importantly, the implications for equine health care and prevention of musculoskeletal injury.

## **Articular cartilage biochemistry**

The hyaline cartilage that covers the articular surfaces of diarthrodial joints is a highly specialised connective tissue with biomechanical characteristics that make it particularly suitable for load bearing and shock absorption. A sparse population of chondrocytes (1-2% of the volume), is distributed throughout the extracellular matrix, which consists mainly of collagen, proteoglycans and water. The physical properties of the tissue depend on the structure and organisation of the macromolecules in the extracellular matrix. The collagen molecules are organised in a dense cross-linked fibrillar network that is packed with proteoglycans which are strongly negatively charged as a result of their polyanionic glycosaminoglycan chains. In this way a large osmotic swelling pressure is created, drawing water into the tissue and expanding the collagen network. It is this balance within the extracellular matrix between the tension in the collagen network and the osmotic swelling pressure of the proteoglycans that gives articular cartilage its unique mechanical characteristics as it provides a combination of high compressive stiffness with a large degree of resiliency. These properties are critically dependent on both the integrity of the collagen network (including cross-linking and lysyl-hydroxylation) and the synthesis and retention of proteoglycans (Kempson, 1980; Eyre and Wu, 1995; Palmer and Bertone, 1996; Todhunter, 1996).

The two major constituents of the cartilage extracellular matrix, collagens and proteoglycans, have very different turnover rates. Turnover time of proteoglycans is variable, probably due to

the existence of sub-populations, and ranges from as little as 3.5 to 1800 days. However, collagen turnover times have been estimated up to 120 years in dogs and 350 years in adult human cartilage (Maroudas, 1980). No specific data are available for the horse, but it can be assumed that the order of magnitude will be similar. It should be explicitly stated that these values apply to mature individuals. In young, growing individuals there is a constant process of remodelling and growth, making turnover rates much higher. The extremely long turnover times in mature individuals are presumably related to the structural features of the mature collagen fibril. Once the collagen fibril has been synthesised and the covalent cross-links (mainly hydroxylsypyrindinoline or HP cross-links, to a much lesser extent lysylpyrindinoline or LP cross-links) have formed within and between molecules in the fibril, the sites available for enzymatic cleavage are limited, giving the triple helical collagen great resistance to proteolytic attacks and hence turning them into rather static tissue components (Maroudas, 1980; Eyre *et al.*, 1991; Maroudas *et al.*, 1992; Todhunter, 1996). It is therefore likely that the notorious and long-known very limited capacity for repair of articular cartilage as alluded to earlier (Hunter, 1743) originates from the virtually absent turnover of the collagen network in mature individuals.

### **Research on extracellular matrix composition and development of articular cartilage in the horse**

The first report on the composition of the ECM of equine articular cartilage dates from the early 90s (Vachon *et al.*, 1990). However, interest in the normal situation seemed to fade quickly as virtually all later biochemical work focused at repair tissue (Barr *et al.*, 1994), or cartilage specimens from specific disorders such as osteochondrosis (Lillich *et al.*, 1997). In some cases normal tissue was investigated together with pathological samples (Todhunter *et al.*, 1994). However, by far most attention was focused at either assessing the effect of a variety of drugs on cartilage metabolism and composition, or on the regulation of cartilage metabolism under physiological and pathological conditions. Studies such as the one characterising the different sub-populations of proteoglycans in different layers (of human femoral head) cartilage (Mitrovic and Darmon, 1994) are totally lacking in equine orthopaedic literature. Site differences in the proteoglycan content of the ECM of equine articular cartilage were, however, reported as a sequel to exercise (Palmer *et al.*, 1995), an environmental factor that was proven to influence proteoglycan metabolism (Little *et al.*, 1997). On a parallel track, Murray *et al.* (1995) demonstrated variations in biomechanical properties of articular cartilage in the midmetacarpal joint, an area that is known to be frequently affected by degenerative joint disease.

In this line, it was suggested that variable loading conditions can only adequately be met (without provoking damage in the long term) by articular cartilage featuring different mechanical properties (Palmer and Bertone, 1996). As biomechanical properties and biochemical composition of tissues are intricately interwoven, this is only possible when there are topographical differences in biochemical characteristics.

Little and Ghosh (1997) were the first to provide some evidence that these topographical differences in extracellular matrix composition may be not yet present at birth, as they demonstrated that in neonatal ovine articular cartilage, in contrast to tissue from mature individuals, there was neither heterogeneity in proteoglycan biochemistry nor in chondrocyte metabolism. This brought them to the hypothesis that the regional chondrocyte phenotype of adult ovine cartilage resulted from factors imposed on the joint after birth, *i.e.* weight bearing and articulation. At that moment the conclusion lay at hand that, if the same principle would

apply to biochemical characteristics of the collagen network which, once established in the mature individual, will not essentially be altered throughout the entire lifespan, it would mean that loading in the early postnatal period could affect the final make-up of the tissue and hence resistance to injury.

In 1999 Brama *et al.*, published a first report on site (and age) related differences in the biochemical characterisation of the collagen network at two sites of the proximal articular surface of the first phalanx (Brama *et al.*, 1999a). They showed a significantly higher collagen content at the dorsal rim of the articular surface than in the central fovea. Numbers of cross-links were higher too. In a more extensive study into the topographical heterogeneity of the same articular surface, in which 12 sites were sampled and proteoglycan content was determined as well, it was demonstrated that there was a distinct and consistent topographical variation for all parameters determined: water, DNA, glycosaminoglycans, collagen, HP cross-links and degree of lysyl hydroxylation (Brama *et al.*, 2000a) (Figure 1). The findings corresponded neatly with the load distribution in the joint as determined using pressure-sensitive films in an *in vitro* setting and applying loads occurring during activities such as standing, walking, trotting, cantering and jumping (Brama *et al.*, 2001). From this study it became clear that there were huge differences in degree of loading throughout the joint. Whereas the central area is loaded during all activities, the dorsal rim is not loaded but during the most strenuous exercise (Figure 2). However, under those circumstances the load is almost double the load in the central fovea. This means that there is a wide scale of differently

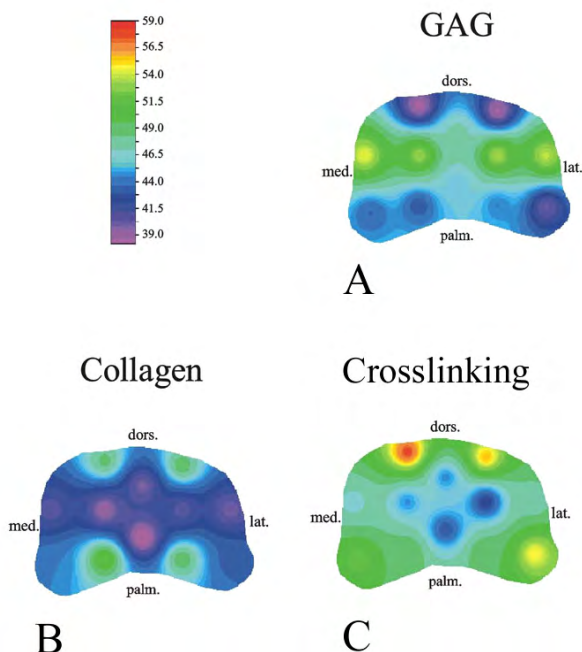


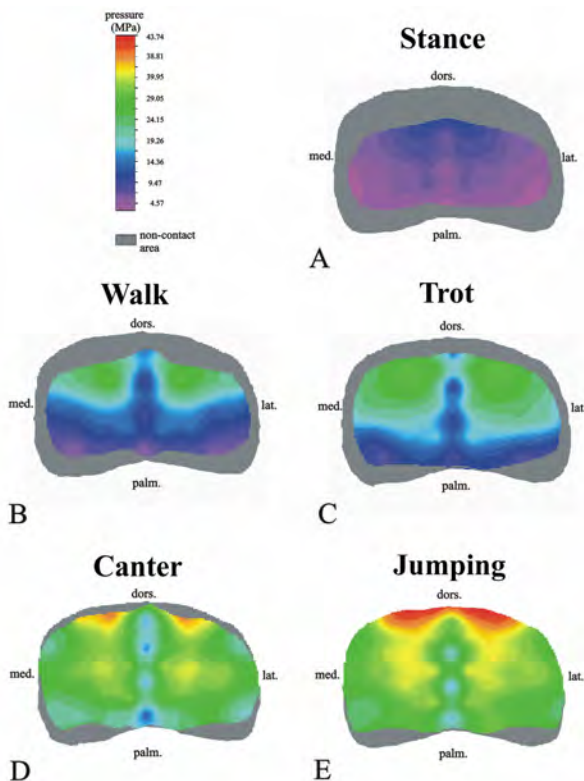
Figure 1. Topographical patterns of measured biochemical parameters on the proximal articular surface of the proximal phalanx of the fetlock joint.

dors.=dorsal; GAG=glycosaminoglycan; Cross-linking = hydroxylysylpyridinoline (HP) cross-links; lat.=lateral; med.=medial; palm.=palmar

(Adapted from: Brama PAJ, TeKoppele JM, Bank RA, *et al.*, Topographical mapping of biochemical properties of articular cartilage in the equine fetlock joint. *Equine Vet J* 2000; 31,19-26).



loaded areas: from sites that are constantly, but relatively mildly loaded, to areas that are rarely, but very heavily loaded. In the latter area collagen content is high, but glycosaminoglycan (GAG) content is low, in the more constantly loaded areas the situation is reverse. This pattern is consistent with findings in human intervertebral discs where Scott *et al.* (1994) demonstrated that increasing compressive loads corresponded with higher GAG-levels while higher collagen levels correlated with greater ranges of torsional and shearing strain. Cross-links are highest at the intermittently, but heavily loaded sites too, presumably in order to withstand the high forces that are generated. Cross-linking is higher medially than laterally, which is likely to be caused by the asymmetric loading in the life animal because of the central, and hence abaxial, position of the centre of gravity. In the *in vitro* drawbench experiment performed by Brama *et al.* (2001) this asymmetric loading was not taken into account. The degree of lysyl-hydroxylation largely mirrors collagen content and cross-linking and thus seems inversely related to biomechanical strength. This is in line with earlier findings in bone where elevated hydroxylysine levels in collagen type I were associated with lower biomechanical strength (Yang *et al.*, 1993; Knott *et al.*, 1995).



*Figure 2. Loading patterns (MPa) and contact areas on the proximal articular surface of the proximal phalanx of the fetlock joint under different loading conditions mimicking various physiological conditions.*

dors.=dorsal; lat.=lateral; med.=medial; MPa=megapascal; N=Newton; palm.=palmar;  
(From: Brama PAJ, Karssen D, Barneveld A, *et al.*, Contact areas and pressure distribution on the proximal articular surface of the proximal phalanx under loading (in the sagittal plane). (Adapted from: *Equine Vet J* 2001;33,26-32)

In an age range of 4-30 years it was demonstrated that the site-related differences (or at least those between the most extremely loaded sites) remained constant throughout life (Brama *et al.*, 1999a). However, it remained yet to be proven that the hypothesis of Little and Ghosh (1997), which was based on studies of ovine articular cartilage, applied to the horse as well (and to other components of the ECM than GAGs alone). A first investigation into the biochemical composition of neonatal equine articular cartilage showed that there were indeed no differences for all biochemical parameters investigated, including those relating to collagen, between the two sites that are most different in the mature animal (Brama *et al.*, 2000b). Site-specific differences had developed at the age of 5 months for DNA, GAGs, collagen and lysyl hydroxylation, but were still absent for water and HP cross-linking at age 11 months. The picture became complete when these data were combined with data from older animals: the ratios between the two sites for these parameters became significantly different from zero in the age span of 1-4 years (Brama *et al.*, 2002) (Figure 3). It is interesting to note that most of the topographical heterogeneity takes shape in the first 5 months of life, which therefore seems to be a crucial period.

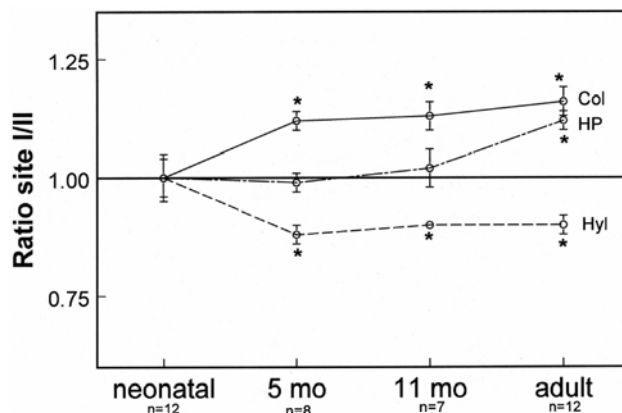


Figure 3. The development of topographical heterogeneity between two differently loaded sites in the same joint (Site I = dorsal margin of proximal articular surface of first phalanx; Site II is central area of the same joint surface). For collagen content (Col) and hydroxylysine level (Hyl), the process of functional adaptation has largely taken place before age 5 months. For hydroxylysylpyridinoline cross-links (HP), developments start later.

Little and Ghosh (1997) presumed that the development of topographical heterogeneity was initiated by the onset of articulation and weight-bearing after birth. In a study on the effect of exercise on the biochemical composition of the articular ECM of young foals, which was part of a much larger investigation into the influence of exercise on the development of the equine musculoskeletal system in general and on osteochondrosis in particular (van Weeren and Barneveld, 1999), Brama *et al.* (1999b) showed that withholding of exercise (box-rest) led to a significantly lower GAG-content than in pastured foals. The difference disappeared when normal exercise was given after the age of 5 months. They were not able to demonstrate changes in collagen parameters between the groups. However, in that study only one site (the central fovea) was examined. Detailed studies into the development of differences in biochemical composition between that area and the dorsal rim of the first phalanx in the same material made clear that in the box-rested foals significant differences in total collagen content and degree of lysyl-hydroxylation had failed to develop at age 5 months (Brama *et al.*, 2002). The most striking finding was that these differences appeared to not develop either

after the foals were subjected to a normal exercise regimen after age 5 months (Figure 4). Apparently, cartilage metabolism had dropped too far to make up for lost ground by that age. The dramatic drop in cartilage metabolism was recently further substantiated by van den Boom *et al.* (2003), who demonstrated an exponential decline in the collagen degradation product hydroxyproline in synovial fluid from foals.

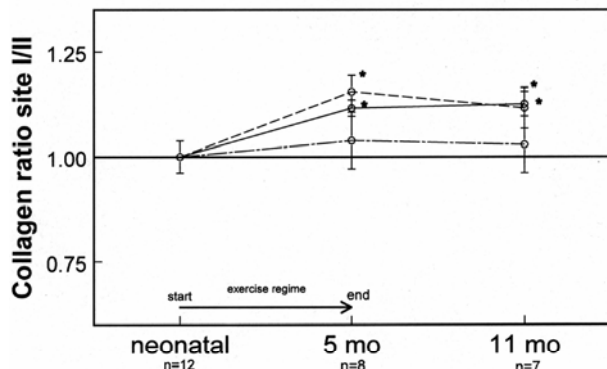


Figure 4. Topographical heterogeneity in collagen between two differently loaded sites (Site I = dorsal margin of proximal articular surface of first phalanx; Site II is central area of the same joint surface) has been developed by age 5 months in two groups of foals that were exercised (--- and \_\_\_\_). This heterogeneity did not develop in a group of foals that was withheld exercise (.....). When this latter group was given additional exercise after age 5 months, the retarded development was not caught up with, probably because of the already insufficient level of cartilage metabolism.

## Osteochondrosis

Support for the vital importance of the early postnatal period with respect to joint development comes from an other area of equine orthopaedic research too. The developmental orthopaedic disease osteochondrosis (OC) has recently been shown to be much more dynamic in character than thought before. After the first indications that lesions may still develop in young animals (Carlsten *et al.*, 1993), Dabareiner *et al.* (1993) showed that small lesions might disappear as well. Later, Dik *et al.* (1999) demonstrated that in the first months of life radiographically visible lesions may come and go, including those in which distinct fragments are present. However, the same authors showed that after a certain age, which varied per joint but did not exceed 12 months, the situation remained static. This research led to the view that discrimination should be made between the pathogenesis of OC lesions and the ensuing repair process. The resultant of both processes determines whether a mature individual will have a lesion or not, *i.e.* final clinical outcome. However, the two processes seem to be very different in nature and may be influenced by different factors. Very recently liver copper content in the foal was shown to affect the repair process of existing lesions, but not the pathogenesis of these (van Weeren *et al.*, 2003). Whereas the real pathogenesis of OC remains largely unknown (Jeffcott and Henson 1998), it lies at hand to presume that the capacity for repair is related to the level of cartilage metabolism. This explains the rapid decline in the maturing individual of the capacity for full repair and the essential cessation of lesion repair at the age of about one year when apparently the “window of opportunity” has definitively closed.

## Discussion

From the recent research a novel concept of the composition and development of equine articular cartilage emerges.

The distinct biochemical heterogeneity as is found in the mature animal is clearly loading-related. Further, the studies on biochemical composition of the ECM in various age classes have demonstrated that at birth a topographical heterogeneity as is characteristic for the mature individual does not yet exist. This heterogeneity is for the largest part formed during the first phase of life. At that moment cartilage metabolism is still high and with it the remodelling rate, which may be related to the fact that up to age 7 months blood vessels can still be found in the ECM of articular cartilage in the form of so-called cartilage canals (Carlson *et al.*, 1995). Van den Hoogen *et al.* (1999) showed that the maximal metabolic capacity in terms of <sup>35</sup>S-incorporation of equine chondrocytes at 11 months of age was more than 30% lower than at age 5 months. It is of utmost importance to note that this process of transformation from biochemical homogeneity to topographical heterogeneity or of functional adaptation as it has been called (Brama *et al.*, 2000b), is to a large part a once in a lifetime process. This process of functional adaptation is not unique to the horse, but is probably common to all mammals and perhaps even all vertebrates. Research in laboratory animals and in man, using different techniques, led to a similar outcome and hypothesis (Helminen *et al.*, 2000).

The consequences are far-reaching. Insufficient exercise at early age may lead to insufficient functional adaptation and hence to a cartilage layer that is less apt to withstand the demands made by athletic performance. The rapid decrease in cartilage metabolism, the loss of vascular supply because of the disappearance of the cartilage canals, and/or the increased density of the collagen network through cross-linking that makes it less accessible for proteolytic enzymes, negatively influence the remodelling rate. These factors give the insufficient functional adaptation or, in case of OC, existing lesions a permanent and irrevocable character. It is not more than logic, though yet unproven, that animals with insufficient functional adaptation are more prone to injury and that they might more easily develop chronic degenerative joint disorders.

These data strongly plea to provide the newly born foal with ample opportunity for exercise. However, care should be taken, as not all types of exercise appear to be beneficial. In the same study as quoted earlier, it was shown that foals that had been kept in a box-stall, but were given additional high-intensity exercise, did indeed not show any retardation in functional adaptation (Brama *et al.*, 2002). However, in culture the metabolism of their chondrocytes could not be stimulated by adding serum, which was interpreted as a kind of exhaustion (Van den Hoogen *et al.*, 1999). As parameters from other tissues indicated similar effects, it was concluded that the combination of box-rest and high-intensity exercise had a deleterious effect (Barneveld and van Weeren, 1999). Recently, studies on protein cleavage products in the serum of these same groups of foals gave further evidence for the negative effect of the combination of forced exercise and box-rest on cartilage metabolism (Billinghurst *et al.*, 2003). So far, unrestricted pasture exercise comes out best.

It is recognised that unrestricted pasture exercise for the new-born foal, though arguably better from a viewpoint of well-being as well, conflicts with the present-day desire for very “early foals”, at least in those areas where climatic conditions preclude full-day turnout early in the year. It is known that nature never planned foals to be born very early in those temperate zones.

Nature may have given us a good argument against the practice of “early foals”: insufficient functional adaptation of articular cartilage may not only affect the well-being of the animal, it may also have negative consequences for performance, or at least longevity, of the mature equine athlete.

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# Thoroughbred growth characterized by a baseline and systematic deviation

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## Introduction

Growth has been described by numerous empirical equations and physiological models. Physiological models are useful for describing growth with future objectives of investigating genetic and environmental influences on growth. The best fitting physiological model is often a flexible sigmoid curve with parameter values that represent biological processes. One difficulty in describing growth is separating the long term sigmoid pattern from short term deviations due to genetics or environment. The objective of this study was to establish a technique for separating weight-age data into two components, a baseline curve and systematic deviation that together may be regarded as a physiological model of growth.

## Materials and Methods

Growth of 175 foals at the Middleburg Agricultural Research and Extension Center was monitored from birth to 16 mo. Mares were bred over a 10 wk period from April 15th to June 22nd, and 95% of foals were born in April and May. Colts were gelded at 1 yr of age in the first and second year and at 3 to 4 wk of age in subsequent years. Foals were weaned gradually, at 6 mo, by removal of two mares from each group every 4 d.

A total of 2,698 body weight records for 175 foals were obtained during a period of 8 yr (1994 – 2001) (Figure 1). Foals were first measured 24 h or 1 mo following birth. Subsequent measurements were taken at approximately 28 d intervals for the following 16 to 19 mo. A sigmoid growth model was fit to the weight-age data (Richards, 1959).

$$W = A(1 \pm be^{-kt})^M \quad (1)$$

The change in BW (W, kg) is described in relation to the change in age (t, d). The sigmoid growth model was fit to the weight-age data using the non-linear mixed procedure of SAS (SAS, 2001). The mixed model had random effects added to both A and M. The addition of the two random effects accounts for within foal variation and different patterns of growth between foals. An improved description of the variance structure also enhances the precision of the other parameter estimates in the model.

Separation of the weight-age data into two components was accomplished in two stages. First, a series of 1,634 sigmoid curves were fit following removal of specified subsets of data with start and end values, such that start = 0 to 540 d by 10 d increments and end = start + 10 d by 10 d increments. Second, the A parameter of the 1,217 curves that converged was plotted on the z-axis of a three-dimensional plot, with the start and end of the removed subsets on the x and y-axis, respectively. Prominent features of the contour plot were evidence of the presence and location of a systematic deviation in the weight-age data. These features were used to accurately identify the data subset with the least influence from the systematic deviation, that is, a baseline data set (BDS).

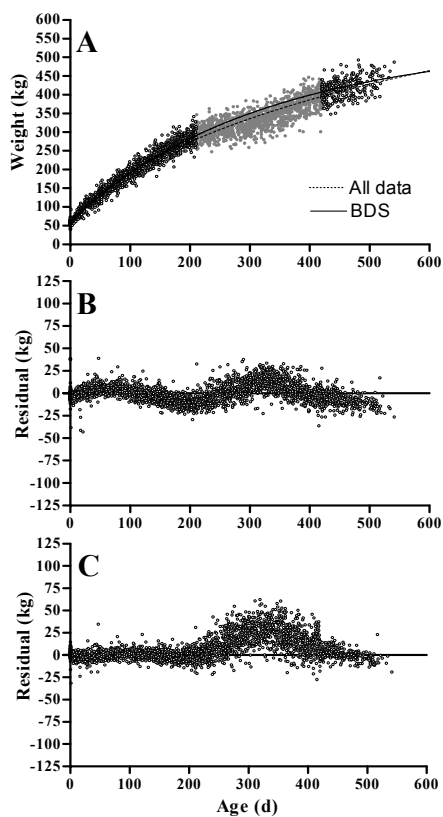


Figure 1. Body weight of horses from birth to 541 d of age (A).  $\circ$  represent data points that are included in the baseline data set (BDS), and  $\bullet$  represent those set aside. Best fit sigmoid curves to both data sets are depicted. Residuals of mixed nonlinear model fit to all the data (B) and mixed nonlinear model fit to the BDS (C).

## Findings

The sigmoid model fit to the BDS indicates a mature weight of  $542 \pm 6.2$  kg reached at 7 yr (Table 1). The residuals from the model fit to the BDS illustrate only one systematic deviation, with a maximum deviation at approximately 325 d of age. The sigmoid model fit to all the data indicates a mature weight of  $752 \pm 22$  kg reached at 20 yr. The residuals from the model also illustrate three possible systematic deviations from the best fit sigmoid curve, with maximum deviations at approximately 200, 325 and 500 d of age.

Table 1. Parameter estimates for mixed sigmoid model's fit to all the data and the baseline data set (BDS).

| Parameter              | A     | b     | k       | M     | Residual<br>variance | Variance<br>(A) | Variance<br>(M) | Covariance<br>(A+M) |
|------------------------|-------|-------|---------|-------|----------------------|-----------------|-----------------|---------------------|
| Mixed model (All data) | 752.4 | 0.988 | 0.00097 | 0.601 | 119.9                | 2590            | 0.0012          | 1.03                |
| Mixed model (BDS)      | 542.2 | 0.919 | 0.00288 | 0.892 | 37.6                 | 1050            | 0.0029          | 0.62                |



The systematic deviation in the weight-age data is characterized by a decrease and subsequent increase in rate of growth that occurs between 210 and 420 d of age. Growth curves from this study as well others illustrate that the deviation is a consistent characteristic of Thoroughbred growth, as there is a maximum deviation from the physiological baseline growth model between 325 and 375 d of age (Hintz *et al.*, 1979).

Fitting the sigmoid function to the BDS yielded parameter estimates that were in agreement with published values. The estimated mature weight was in better agreement with the estimates of Willoughby (1975). The residuals illustrate only one systematic deviation in the data versus at least three seen fitting the model to all the data. The *M* parameter ( $0.89 \pm 0.02$ ) is significantly different from one ( $P < 0.0001$ ) illustrating the necessity of using a sigmoid function that does not assume a value for *M*.

This study applied a goodness of fit procedure to differentiate between a systematic deviation in weight-age data and a baseline curve, hence enabled development of a physiological model of growth in Thoroughbreds. This novel procedure could be applied to other growth scenarios. A systematic deviation was identified from 210 to 420 d and was attributed mainly to changes in the environment. The baseline curve predicted mature weights in line with the literature, about  $542 \pm 6.2$  kg. Improved characterisation of growth should lead to advances in determining nutrient and energy requirements, as well as the development and testing of novel feeding management practices that promote desired growth rates.

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# The effect of age of dam on birth weight and growth rate of Thoroughbred foals

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## Introduction

Thoroughbred (TB) growth rate data has been documented under various environmental and management scenarios. Hintz *et al.* (1979) published data of TB foal growth over an 18-year period on a single Canadian farm. Thompson (1995) and Pagan (1998) measured growth of TBs that were raised in commercial management regimes in Kentucky. The following data were collected on a weekly basis on a commercial TB breeding farm in Midway, KY. The objectives of this study were to determine if 1. Foals from aged broodmares were lighter at birth, 2. The growth rate of those foals differed from other foals, 3. If growth rates of foals that were fostered on nurse mares varied from own-dam foals, 4. If there were gender differences in birth weights or growth rates, and, 5. If weekly foal weight measurements were necessary to document variations in TB growth patterns.

## Materials and methods

Fifty-six TB foals (28 fillies and 28 colts) were weighed over a 9-month period on a weekly schedule using a Tru-test Agriculture Weighing System (700 Series) electronic digital readout scale. Foals were weighed from mid-February to two weeks prior to shipment to the Keeneland November Mixed TB Sale. Fourteen of the foals were fostered on to nurse mares within 24-48 hours of birth. Foals were offered alfalfa hay and creep fed a mixed grain ration with dams two times daily. Mare/foal pairs were turned out daily in small groups (2-5 mare/foal pairs) and had free access to water and trace mineralized salt. Post weaning, foals were managed under the same conditions (Willard, 2003).

Birth weight data was analyzed using mixed models analysis of variance (Proc Mixed, Littell *et al.*, 1996) with individual foal as a random effect. Effects of gender and age group of dam ( $\leq 14$  years, 15-19 years, and  $\geq 20$  years) on birth weight were tested separately. Body weight data for growth rate determination was analyzed using a linear mixed models approach with age as a continuous variable and individual foal as a random effect (Proc Mixed, Littell *et al.*, 1996). Effects of gender and age group of dam were tested as discrete variables. Body weight data was also fitted to the logistic equation using Proc NLMixed (Wolfinger, 1999). Prediction equations from linear and non-linear regression were compared.

## Results and discussion

### Dam age effect on birth weight

Using orthogonal comparisons, old mares ( $\geq 20$  years) had lighter foals ( $p < 0.1$ ) than the other groups. The average foal birth weight was 4 kg less than foals from mares 19 years of age or younger. There was no significant difference of birth weight due to gender across the data set.

### Dam age effect on foal growth rate

Foals from dams ( $\geq 20$  years) grew significantly faster (0.07 kg/day) than foals from other groups. However, eleven out of fourteen foals in this group were on nurse mares. Therefore, growth data of foals from dams twenty years or greater was confounded with foals raised on nurse mares. Also, no differences were observed for growth rates of colts vs. fillies.

### Nurse mare effect on foal growth rate

When data from foals fostered by nurse mares ( $n=14$ ) were compared to own-dam raised foals ( $n=42$ ), fostered foals grew significantly faster (0.0366 kg/day) than own-dam raised foals ( $p < .0002$ ). Most nurse mares were Light Breed (primarily purebred and crossbred Tennessee Walking Horses) with an average weight of 900 pounds and age of 14 years (Willard, 2003). Unlike TBs, which are selected for performance, nurse mares are specifically selected based upon reproductive, mothering, and milk production qualities.

### Comparison to previous KY TB growth data (Pagan, 1998)

The non-linear equation did not result in an improved fit for this data as compared to the linear equation, which is expected given that the growth rates are being determined over a short period of post-natal growth (up to 10 months of age). The equation matches previous data reported for Kentucky-bred TB foals (Pagan, 1998).

## Conclusions

In this population, older mares produced smaller foals at birth, but nurse mares helped them grow faster and catch up to their peers. This population of highly managed foals raised under commercial conditions for the intention of marketing them as weanlings grew at consistent rates with Pagan (1998). Based upon the fact that there was more variation within the population versus the week-to-week variation within an individual, bimonthly measurements of foals should be adequate to determine overall growth rate curves.

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# Growth patterns in Thoroughbred horses during the second year of life

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## Introduction

Many studies have been conducted which examine the growth of Thoroughbred horses from birth through 18-24 months of age (Thompson *et al.*, 1994, Jelani *et al.*, 1996; Pagan *et al.*, 1996), but there are a few informations after this period, particularly during training period.

## Materials and methods

Over a five year period (1999-2003), a total of 131 Thoroughbred were weighed every three months from the beginning of training through 34 months of age. Withers height, chest and cannon bone circumferences were also measured. All horses were stabled at San Rossore Training Center (Pisa) and were trained regularly during the considered period. The animals were fed according I.N.R.A. requirements (1990).

## Results

The results showed a significant increase in all the measurements considered (Table 1, Figure 1 and 2). Moreover data showed significant differences in growth patterns of weight and cannon circumference between male and female (Figure 3 and 4). Male had a larger ( $p<0.01$ ) cannon bone with a  $20.05\pm0.67$  cm circumference than the female, with a  $19.46\pm0.56$  cm circumference at the end of the considered period. Consequently body weight resulted significantly ( $p<0.01$ ) higher in male than in female ( $476\pm34$  versus  $444\pm35$  respectively). No differences were recorded in withers height and chest circumference between sexes.

Table 1. Growth data from about 22 to 34 months of life (mean $\pm$ SD).

| Months of age       | 22                 | 25                 | 28                 | 31                 | 34                 |
|---------------------|--------------------|--------------------|--------------------|--------------------|--------------------|
| Body weight (kg)    | 425.67 $\pm$ 27.62 | 441.53 $\pm$ 29.90 | 449.21 $\pm$ 30.31 | 460.17 $\pm$ 34.48 | 459.52 $\pm$ 36.81 |
| Withers height (cm) | 155.41 $\pm$ 3.51  | 157.45 $\pm$ 3.65  | 159.90 $\pm$ 3.20  | 159.65 $\pm$ 3.18  | 160.54 $\pm$ 3.33  |
| Chest cirf (cm)     | 174.43 $\pm$ 3.69  | 176.76 $\pm$ 3.72  | 178.42 $\pm$ 3.68  | 179.58 $\pm$ 3.44  | 180.99 $\pm$ 3.84  |
| Cannon cirf (cm)    | 19.29 $\pm$ 0.62   | 19.47 $\pm$ 0.62   | 19.65 $\pm$ 0.65   | 19.69 $\pm$ 0.59   | 19.78 $\pm$ 0.65   |

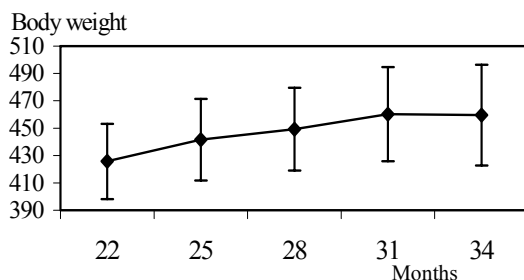


Figure 1. Body weight through the period (mean  $\pm$  SD).

Wither height

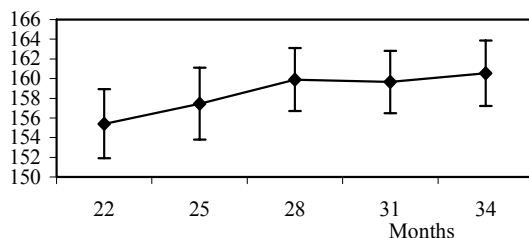


Figure 2. Wither height through the considered period (mean  $\pm$  SD).

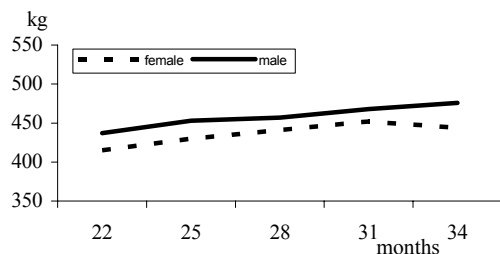


Figure 3. Growth pattern of body weight in male and female.

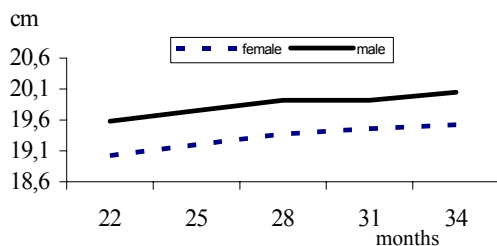


Figure 4. Growth pattern of cannon bone in male and female.

The obtained data show a further growth during the first period of training and racing. For this reason a correct management for a normal growth pattern of Thoroughbred horses is important.

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# Percentiles: A simple tool to monitor the growth of Thoroughbreds

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## Introduction

Growth rates of thoroughbreds are a useful aid to check the correct muscle and skeletal development in growing horses, in order to prevent developmental orthopaedic diseases.

At present few studies are available regarding Italy (Cicogna *et al.*, 1991). The most relevant contributions come from American authors (Hintz *et al.*, 1979; Thompson, 1988; Thompson, 1995; Pagan, 1996; Ott and Asquith, 1986). Therefore we have studied the growing patterns of colts and fillies, from 5 to 20 months of age, raised in some studs in Northern Italy in order to define a simple tool, easy to use and to monitor growth. The percentile curves obtained are descriptive statistics also useful in detecting diverging growth which could lead to skeletal abnormalities in growing horses.

## Materials and methods

On a total of 354 thoroughbreds raised in 5 Italian studs in Lombardy and Piedmont, 171 fillies and 183 colts were studied under traditional management conditions. Body weight, wither height, thorax circumference and cannon bone circumference were monitored from 1990 to 1999 for a total of 10,494 records. All measures were recorded every month, at about 30 day intervals, from 5 to 20 months of age, with the exception of the cannon bone circumference recorded every two months. Body weight was not available for one stud. Growth regression models for each somatic measurement, separately for colt and fillies, were calculated at different ages. The data have been summarized with the percentile analysis, calculating the value of the 10<sup>th</sup>, 25<sup>th</sup>, 50<sup>th</sup>, 75<sup>th</sup>, and 90<sup>th</sup> percentile between 9 and 18 months of age. The analyses here shown are limited to weight regression curves of colts and fillies, to weight percentiles of colts and to an applicative percentile example on withers height.

## Findings

Weight regression curves are shown in Figure 1 and the respective equations with the relative determination coefficients, for fillies and colts, are the following:  $y(kg) = 96.18 + 0.82 \times age(days) - 0.0004 \times age^2(days)$  with an  $r^2=0.76$ ; and  $y(kg) = 123.68 + 0.70 \times age(days) - 0.0002 \times age^2(days)$  with an  $r^2=0.74$ . Moreover, concerning withers height we registered, respectively at 8, 12 and 18 months of age, for fillies and colts, the following mean value ( $\pm$ S.E.): at 8 months, 138.69 $\pm$ 0.66 cm and 139.34  $\pm$  0.65 cm; at 12 months 145.39  $\pm$ 0.31 cm and 147.11 $\pm$ 0.33 cm; at 18 months 153.96  $\pm$ 0.57 and 154.85  $\pm$ 0.55 cm. Italian weights and withers height were always higher for colts than for fillies in accordance with the above-cited American literature.

Percentile curve weights for colts, shown in Figure 2, can be a useful tool to monitor growing thoroughbreds. Growth should be regular and follow the percentile lines. In the withers height

percentiles shown in Figure 3, an example of a colt growth pattern is reported. This colt shows an irregular growth at withers height especially from 14<sup>th</sup> to 17<sup>th</sup> months of age, this trend could be related to nutritional imbalances. With this tool it is easy to identify anomalous growth. Therefore it is possible to intervene to correct possible dietary errors especially energy and protein intakes or other environmental factors known to influence growth. Diet composition and pasture availability should be controlled when irregular growth is evidenced, especially if several measures are implicated.

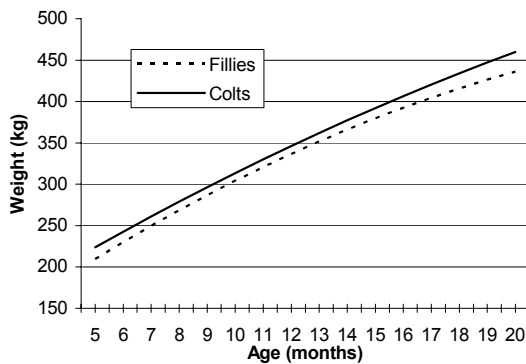


Figure 1. Weight regression curves of thoroughbred colts and fillies.

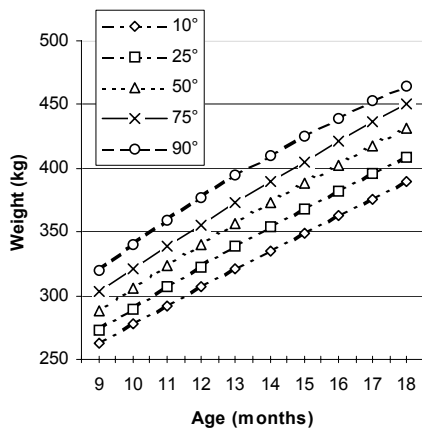


Figure 2. Weight pattern percentiles in colts.



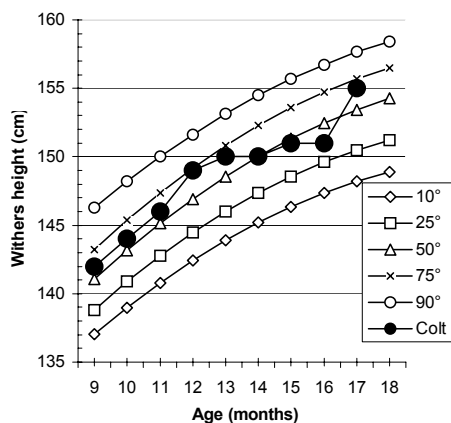


Figure 3. Withers height percentile pattern in colts: an example.

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# Predicting growth rates and mature sizes in Morgan horses

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## Introduction

Lawrence *et al.* (2003) evaluated growth rates of Morgan horses by pooling data points of post-weaning foal weight measurements from the University of Vermont (UVM) Morgan Horse Farm from 1965-1997. A polynomial equation [ $y = 0.0004x^2 + 0.1141y + 175.07$ ; where  $y$  = weight (kg) and  $x$  = age (d)] was developed to predict Morgan weights between 150-540 days of age. The following data were collected over the past five years at the UVM Morgan Horse Farm. The objectives of this study were to 1. Compare linear and nonlinear equations in order to establish a mature weight prediction equation for the data set, 2. Determine if foal gender or age of dam affected birth weights and growth rates of foals, 3. Challenge the polynomial prediction equation (Lawrence *et al.*, 2003) using current data from individual horses, and 4. Compare the growth rates of Morgan and Thoroughbred horses (Pagan, 1998).

## Materials and methods

Fifty-nine Morgan foals (35 fillies and 24 colts) from five foal crops (1998-2002) were weighed post-weaning on a bi-weekly schedule using a Howe Scale Company Single Beam Portable Platform Scale. Foals were group-housed in dry lot pens with shelters in groups of 4-8 animals per pen and fed a mixed grain diet with grass hay twice daily. Foals had free access to water and trace-mineralized salt.

Birth weight data was analyzed using mixed models analysis of variance (Proc Mixed, Littell *et al.*, 1996) with individual foal as a random effect and gender as a fixed effect. Body weight data for growth rate determination was analyzed using a linear mixed models approach with age as a continuous variable, individual foal as a random effect, and gender as a fixed effect (Proc Mixed, Littell *et al.*, 1996). Linear equations were also developed using the same model for foals less than 1 year of age, 1 to 2 years old, and more than 2 years old. Body weight data was also fitted to the logistic equation using Proc NLMixed (Wolfinger, 1999). Prediction equations from linear and non-linear regression were compared.

## Results and discussion

### Linear versus nonlinear equations for Morgan growth curve predictions

A linear equation of growth over the period from weaning to more than two years old resulted in a very poor fit. Better fits were obtained by developing equations for groups < 1 year, 1-2 years, and > 2 years of age. However, these linear equations also showed a bias of over predicting body weight during the younger half of the age group and under predicting body weight during the older half of the age group and consequently underestimating growth rates over the entire period.

The non-linear logistic equation resulted in a much-improved fit with unbiased residuals as compared to linear equations of growth.

$$bw = \frac{410}{1 + \exp(-0.0054 \times (age - 190))} \quad (bw = \text{body weight (kg)})$$

The predicted asymptote of 410 kg is lower than 450 kg mature body weight of stallions and unbred mares at the UVM Morgan Horse Farm (S. P. Davis, 2003). The parameter  $b_3$  corresponds to the age at which growth rate starts declining, and was predicted to be 190 days, or approximately one to two months post-weaning. The logistic equation provides a better fit to the data than the polynomial equation reported in Lawrence *et al.* (2003), which was limited to approximately 150 to 540 days of age. It also could be used to predict mature body weight based on post-weaning growth.

### **Dam age effect on birth weight and foal growth rate**

Gender had no significant effect on birth weights. However, foals born to dams 15-years or older were 1.5 kg larger at birth ( $p < .0001$ ), but grew at a slightly slower rate. This may be due to other causes than age of dam, such as genetics.

### **Comparison to previous Morgan model and ky thoroughbred growth data**

When the current Morgan data was plotted against previously derived equations (Lawrence, *et al.* 2003), the nonlinear logistic equation provided a more accurate model for predicting Morgan growth rates and mature weights. The increased prediction accuracy may be reflective of individual horse data, as compared to the pooled data over a 32-year span (Lawrence, *et al.*, 2003). This population of foals, raised for the intention of marketing as mature show horses, grew much slower (0.59 kg/d vs. 1.95 kg/d) than Thoroughbreds (Pagan, 1998).

### **Conclusions**

Growth prediction curves for this population of Morgan foals are best described using a nonlinear logistic equation. Older mares produced foals of greater birth weight, but those foals grew slower than the rest of the population. Fillies out paced colts in year one, but there were no significant differences between genders in subsequent years, nor in mature body weight as predicted by the logistic equation.

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# First results from a morphological approach to draught foal and filly growth in the Ardennes: 1. Evolution as a function of age

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## Introduction

The growth of the Ardennes horse is not very well known as compared to other breeds<sup>1</sup>. Biometry, the scientific method allowing the study of measurable characteristics through the use of mathematics and statistics, was largely used in the past for draught horses. Currently, that method seems obsolete. However, biometry may still allow us to assess growth and can help detect skeletal defects as well as determine, for instance, the horse's athletic abilities. The purpose of our presentation is to report and compare different measurements made on 32 draught foals from the Ardennes, in order to evaluate their work capacity.

## Materials and methods

A set of 32 pureblood Ardennes foals – 18 males and 12 females – registered in the Belgian studbook of Ardennes draught horses, were placed in standardised conditions. They were 8 months old at the beginning of the study. They received a composite foal food ration at the rate of 1 kg per 100 kilos of live weight, as well as hay. They had access to a meadow, which functioned as a paddock. Various morphological parameters were measured for all foals once every 7 weeks. The measurements included: weight, height up to the withers, height up to the hindquarters, neck length, front limb length, front cannonbone circumference, pastern length, thoracic perimeter, chest width and body length. The animals' average age was 249 days at the first round and 557 days at round 7.

## Results and discussion

The results this presentation is based on were obtained through the first 7 rounds of measurement, which correspond to one year. A total of 191 weighing operations and 223 measurements were available for the 321 foals. Figure 1 gives the evolution of withers size, hindquarter height, body length, and chest width, as a function of age. All these parameters show an evolution, as a function of time, of a sigmoid kind, with an inflexion point corresponding to the time of puberty, it being a period of intense growth occurring usually round the month of April after the horse's first year of age. The phase of intense growth occurs at different times during puberty, depending on the parameter observed. It takes place round the 10<sup>th</sup> month for the body length. For wither size, hindquarter height, neck length, front limb length, and cannonbone circumference, intense growth takes place round the age of one. On the other hand, for the weight, chest width, and the pasterns' length, faster growth occurs round the age of 14 months. Although the sigmoid shape is evident on the curbs, the measurements were taken during a relatively linear growth phase. Towards the end of the

period under study, corresponding to the age of 18 months, the growth speed begins to diminish (Green 1976). Normally, growth should become slower only when the animal is about 2 years old (Martin-Rosset, 1990). Sex has a highly significant influence on the height, chest width, neck length, front limb length and cannonbone circumference. A significant correlation may also be observed between the sex and the hindquarters' size on the one hand, and between the sex and the pasterns' length on the other. Weight, body length and thoracic perimeter bear no correlation to the foal's sex. Although differences due to sex are only significant with regards to certain parameters and are usually rather minimal, the values observed for foals are most often higher than those observed for fillies. There is only one case where the females' measurements are higher than the males': body length at the 7<sup>th</sup> round, where females were on average 0.8 cm longer than the males. Differences between males and females become undoubtedly more pronounced during the following rounds, with males developing more. According to Martin-Rosset (1983), sexual dimorphism becomes obvious only when the animals are 18 months old.

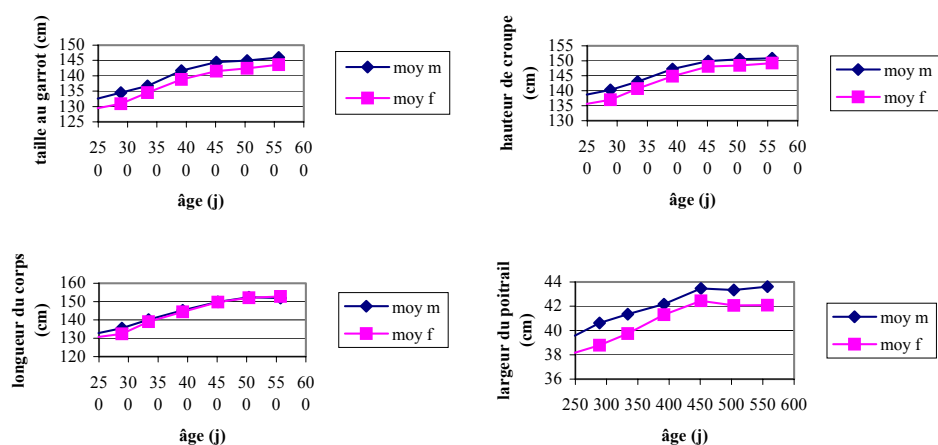


Figure 1. Evolution of the withers, hindquarter height, body length and chest width as a function of age.

## Conclusion

The carrying out of the morphological evaluation protocol allows us to obtain a complete report on the animal's morphology. The above-presented work should serve as a set of tools for a complete study of the growth period of Ardennes foals. Data were collected until the age of 30 months. These data are currently being processed. The protocol followed may be applied to breeding foals, which may help draw conclusions on the best possible breeding conditions or suggest new ones.

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## First results from a morphological approach to draught foal and filly growth in the Ardennes: 2. Calculating growth curbs

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### Introduction

The growth of the Ardennes horse is not very well known as compared to other breeds'. Various biometric parameters may be found in the previous paper. It seemed useful to continue using the obtained data to make up various growth curbs on the basis of the weighing operations carried out during the measuring periods designated as "measurement rounds".

### Materials and methods

Animal description and measurements were given in the previous paper. Descriptive statistics were worked out for each parameter and each round using the SAS *Univariate* procedure. Various linear and non-linear models were adapted using the SAS REG and NLIN procedures as applied to the study on bovines carried out by de Behr *et al.* (2001).

### Results and discussion

Table 1 gives the equations for male growth for the various models. Determining coefficients varied between 0.655 and 0.627 (Table 1) and were not significantly different from one another.

Table 1. Equations for male growth;  $x$  = age (d) and  $y$  = weight (kg).

| Models          | Equations                                                                         | R <sup>2</sup> |
|-----------------|-----------------------------------------------------------------------------------|----------------|
| Segment         | If $x < 425$ : $y = 115.6 + 0.901x$ et $x \geq 425$ : $y = 379.5 + 0.315$         | 0.655          |
| Cubic           | $Y = 205.1 - 0.17x + 0.004x^2 - 4.41 \cdot 10^{-6}x^3$                            | 0.655          |
| Quartic         | $Y = -1228.1 + 14.301x - 0.050x^2 + 8.2 \cdot 10^{-5}x^3 - 5.11 \cdot 10^{-8}x^4$ | 0.655          |
| Quadratic       | $Y = -108.1 + 2.168x - 0.002x^2$                                                  | 0.654          |
| Gompertz        | $Y = 608.5 \exp(-3.178 \exp(-0.0065x))$                                           | 0.652          |
| Brody           | $Y = 628.5 (1 - 1.6472 \exp(-0.0048x))$                                           | 0.652          |
| von Bertalanffy | $Y = 614.2 (1 - 0.8498 \exp(-0.0059x))^3$                                         | 0.652          |
| Exponential     | $Y = 628.5 - 1035.2 \exp(-0.00483x)$                                              | 0.652          |
| Root            | $Y = -88.506 + 27.83x^{0.5}$                                                      | 0.637          |
| Linear          | $Y = 194.067 + 0.676x$                                                            | 0.627          |

Table 2 gives the most probable weight for a specific age using the equations from Table 1. Some of these data (negative weight or exaggerated values) seem completely impossible. Hence, two models have been retained to describe the evolution of weight as a function of age.

*Table 2. Predicted weight of males based on various equations.*

| Models          | Weight<br>at day 0 | Weight<br>at 9.5 months | Weight<br>at 18 months | Adult Weight<br>(5 years) |
|-----------------|--------------------|-------------------------|------------------------|---------------------------|
| Segment         | 115.6 kg           | 375.9 kg                | 554.9 kg               | 954.6 kg                  |
| Cubic           | 205.1 kg           | 374.4 kg                | 555.2 kg               | -13954.7 kg               |
| Quartic         | -1228.1 kg         | 376.6 kg                | 569.8 kg               | -209088.7 kg              |
| Quadratic       | -108.1 kg          | 372.2 kg                | 556.4 kg               | -1980.6 kg                |
| Gompertz        | 25.3 kg            | 372.3 kg                | 557.8 kg               | 608.5 kg                  |
| Brody           | 221.7 kg           | 527.8 kg                | 600.9 kg               | 628.4 kg                  |
| Von bertalanffy | 25.4 kg            | 372.0 kg                | 557.7 kg               | 608.6 kg                  |
| Exponential     | -406.7 kg          | 372.2 kg                | 558.2 kg               | 628.3 kg                  |
| Root            | -88.5 kg           | 384.5 kg                | 568.2 kg               | 1100.2 kg                 |
| Linear          | 194.1 kg           | 389.6 kg                | 570.8 kg               | 1428.6 kg                 |

The first model was the segment one. It was classified as one of the five best models for the various morphometric parameters. The first reason why this model was kept lay in the fact that it was easy to understand and to use. This model gives two linear equations. The first equation applies the parameter to foals 0 to 425 days of age, and the second to foals older than 425 days. On the other hand, for the period under study, the estimation of the morphology's parameters based on these equations was accompanied by a small deviation from the average. Moreover, the estimates of the various parameters carried out using this model are realistic. Its one downfall lies in its reliance on graphical observation, which may be inaccurate, when choosing the abscissa of equation change.

The second model retained was the Gompertz one. It has indeed been classified as one of the five best models (the highest 5  $R^2$  without any significant differences) for the various morphometric parameters. Moreover, it allows for a good estimate of the adult value for the applied parameter.

## Conclusion

The models retained to describe the growth curves of Ardennes foals were: the Segment model; because it was easy to understand and use, and the Gompertz method, which rendered more precisely the sigmoid shape of growth curves.

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# Effect of nutrition on the growth curve of weanling foals

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## Introduction

Nutritional factors are known to influence growth and development of foals. Weanlings are sensitive especially to the quality of the dietary protein (Ott *et al.*, 1981, Saastamoinen and Koskinen, 1993). The aim of the present study was to compose growth curves for normal growth of Finnhorse foals from weaning to the age of one year, and to examine the influence of nutrition on their growth. Weight and feeding data were collected from feeding experiments carried out at MTT Equine Research Centre. The total number of animals was 78.

## Results

Mean daily energy and protein intakes of the foals were 63.4 MJ ME and 494 g DCP, respectively. Lysine intake was approximately 41 g/d. The energy and protein intakes were slightly lower than recommended (64.4 MJ ME and 550 g DCP) for weanlings (*Finnish Feeding Tables*), but the average lysine intake was sufficient (recommended 36 g/d).

### Growth of the foals

The average weight gain of the foals was 507 g/d, varying from 612 g/d at the age of 7 months to 397 g/d at the age of one year. Weight at different ages was increasing from average 267 kg at the age of 7 months to 361 kg at the age of 13 months. Consequently, the weight curve was increasing at the same time when the weight gain curve was decreasing, which is a normal tendency (Figure 1). The affect of age on the weight gain was statistically significant ( $p < 0.001$ ).

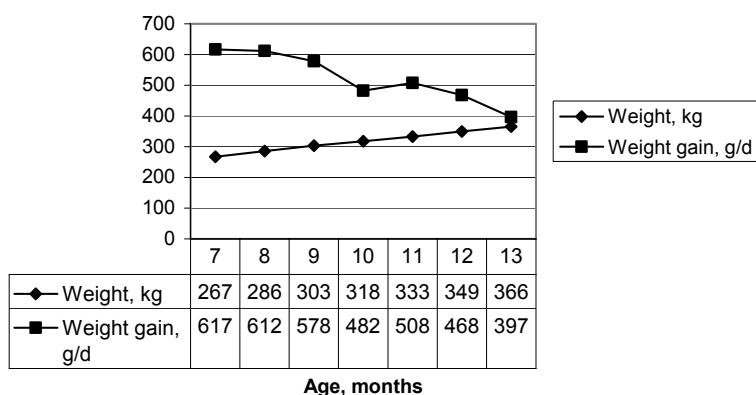


Figure 1. Mean growth and weight curves.

## Effect of the feeding

The energy intake affected on weight gain statistically significantly ( $p<0.01$ ) and positively. The effects of the protein or lysine intakes on growth rate were not statistically significant, however the growth curves between the sufficient (at least 36 g/d) or insufficient (less than 36 g/d) lysine intakes were different (Figure 2).

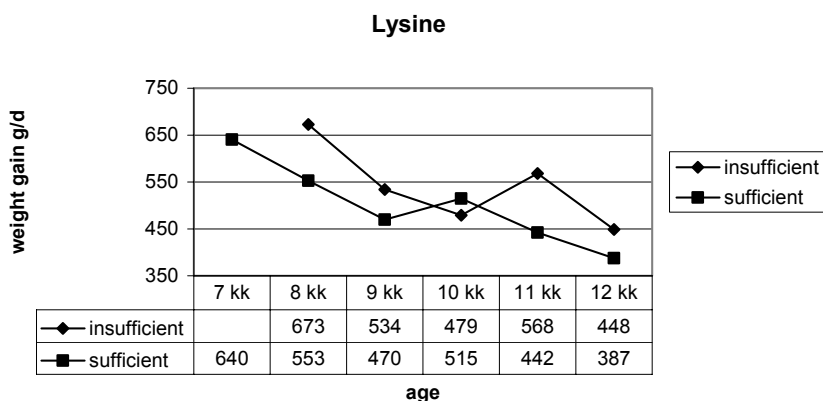


Figure 2. Weight gain according to the lysine intake.

Energy ( $p<0.001$ ), protein ( $p<0.05$ ) and lysine ( $p<0.001$ ) intakes had a statistically significant effect (regression) on weight at different ages. The effects of energy and lysine intakes were positive, and the effect of protein intake was negative, indicating that when energy or lysine intake increases, weight also increases, but when protein intake increases, weight decreases. The sex of the foal had no effect on weight or weight gain.

## Conclusion

The growth rate was influenced by the age, delaying smoothly when the foals became older. Weanling foals are sensitive to the quality of the dietary protein as reported also in previous studies (e.g. Saastamoinen and Koskinen 1993), lysine being the first-limiting amino acid for growth. The protein requirement of foals is smaller when good-quality protein is fed, and excess dietary protein reduced growth as stated also by Yoakam *et al.* (1978). This is due to the fact that excess protein may cause additional energy costs due to extra heat production because of the catabolism of excess amino acids. In this study, the energy intake was lower than recommended. If growth is reduced by restricting energy intake, this may result in compensatory growth later, generally on pasture.

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# The influence of mineral supplementation on the growth of Lusitano foals

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## Introduction

Growth and skeletal development in horses are mainly functions of genetic and nutrient availability (Trillaud-Geyl *et al.*, 1986; Bigot *et al.*, 1987; Ott and Asquith, 1989; INRA, 1990).

The diets traditionally used in the Lusitano horse breed are very often imbalance in the mineral composition, mainly in what concerns Ca/P ratio. The objective of this study was to determine whether the level of Ca, P and Mg in traditional diets (oat and poor hays) vs. supplemented diets, influence growth in Lusitano foals from weaning to one year old, as assessed by body measurements.

## Materials and methods

### Animals and data collection

Two homogenous groups of eight colts each (258 d average age), received for 140 days iso-energetic and iso-proteic diets, that differed only on Ca, P and Mg levels (Table 1).

The basal diet consisted of traditional feeds (oat, horse bean and oat hay) to provide INRA (1990) recommended nutrient allowances for moderate growth. Both groups (A and B) were supplemented with trace minerals and vitamins, but only group B was supplemented with Ca, P and Mg.

Table 1. Individual average daily nutrient intake of the foals in A and B groups.

| Group | UFC  | MADC<br>(g) | Ca<br>(g) | P<br>(g) | M<br>(g) | MS<br>(kg) | Cu<br>(mg) | Zn<br>(mg) | Vit.A<br>(UI) | Vit.D<br>(UI) | Vit.E<br>(UI) |
|-------|------|-------------|-----------|----------|----------|------------|------------|------------|---------------|---------------|---------------|
| A     | 4.76 | 470         | 19.1      | 20.5     | 9.1      | 7.4        | 79.5       | 421        | 45520         | 14350         | 122           |
| B     | 4.76 | 470         | 41.3      | 32.1     | 13.1     | 7.4        | 79.5       | 421        | 45520         | 14350         | 122           |

The animals (8+8) were housed in two 150 m<sup>2</sup> covered parks. All foals received a diet with no macro-mineral supplementation for a 15 day adaptation period.

Periodically (0, 35, 70, 105 and 140 days), the animals were weighed and measured (with height, hip height, girth and cannon circumference) for body growth evaluation.

## Statistical analysis

To evaluate whether data were normally distributed, Shapiro-Wilks and Kolmogorov-Smirnov tests were carried out. All variables under study had a normal distribution. The time was considered as a co-variable and the linear and quadratic effect of the time and the interaction treatment\*time was tested in each case. Analyses were performed with SAS by GLM procedure.

## Results

No significant differences were observed between treatments, with respect to weight or other body measurements. Throughout the experiment, weight and girth presented a linear evolution, while wither height, hip height and cannon circumference presented a quadratic evolution (Table 2).

Table 2. Models of weight and body measurements evolution from day 0 to day 140.

| Groups | Measure                   | Model                                                                                    | R <sup>2</sup> | CV    | P      |
|--------|---------------------------|------------------------------------------------------------------------------------------|----------------|-------|--------|
| A<br>B | Weight (kg)               | YA=251.938+0.498*t<br>YB=244.032+0.498*t                                                 | 0.965          | 2.50% | 0.0001 |
| A<br>B | Girth (cm)                | YA=144.218+0.081*t<br>YB=145.499+0.081*t                                                 | 0.919          | 1.65% | 0.0001 |
| A<br>B | Wither height (cm)        | YA=134.662+0.114*t-0.00035*t <sup>2</sup><br>YB=135.156+0.114*t-0.00035*t <sup>2</sup>   | 0.927          | 1.04% | 0.0001 |
| A<br>B | Hip height (cm)           | YA=140.155+0.081*t-0.00024*t <sup>2</sup><br>YB=140.64+0.081*t-0.00024*t <sup>2</sup>    | 0.897          | 1.04% | 0.0001 |
| A<br>B | Cannon circumference (cm) | YA=17.353+0.0139*t-0.000046*t <sup>2</sup><br>YB=17.222+0.0139*t-0.000046*t <sup>2</sup> | 0.757          | 2.25% | 0.0001 |

t = 0, 35, 70, 105, 140 day

## Discussion

No significant effects were observed on foals growth rate or body conformation for both treatments. The same fact have been reported by Schryver *et al.* (1974) and Jordan *et al.* (1975) with different horse breeds and different Ca and P levels.

Linearity of growth was referred by Bigot *et al.* (1988) for “Selle Français” and Anglo-Arab foals between 9 and 18 months old, which was confirmed in this study concerning weight and girth circumference. The animals gained an average of 498 g (±16) from weaning to one year old what is in agreement with the study reported by Bigot *et al.* (1987) for a moderate growth.

Hip height remained slightly larger than wither height, throughout the study. This observation has been already made by Thompson (1995) in the same aged Thoroughbred foals.

The fact that Ca, P and Mg levels in the basal diet were close to supply the animal needs, could explain the observed results. However, this animals should be followed in the future. The assessment of bone mineral status should be considered in further studies.

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# Growth rates in Hanoverian Warmblood foals and the development of osteochondrosis

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## Introduction

Rapid growth in foals is associated with a higher risk of osteochondrotic lesions. To date, there is only limited new information about the optimal growth rate in Warmblood foals. Available data usually encompass only a small number of foals and may not reflect growth rates under normal conditions and the occurrence of osteochondrotic lesions. The presented study was performed to obtain information on growth rates and the incidence of osteochondrotic lesions in Hanoverian Warmblood foals under typical field conditions. This investigation was part of a larger project in which feeding, housing, and genetics were evaluated with special reference to the development of osteochondrosis in foals.

## Material and methods

629 Hanoverian Warmblood foals born between December 26, 2000 and July 01, 2001 (308 males, 321 females) from 83 farms in Germany were included in this study. Over a period of six months foals were weighed monthly on a portable electronic scale. The withers height was measured at the highest point of the withers using a standard measuring stick. Furthermore feeding practice and the level of nutrient intake (DE, DCP, major minerals and trace elements) of the mares and the foals were evaluated during this observation period.

Information on osteochondrosis (OC) was obtained between the fifth and tenth month after birth using x-ray (fetlock joints, hocks, and stifles). The effects of sex, birth month, and growth rates on the incidence of OC were tested by analysis of variance. All results are presented as means  $\pm$  SD.

## Results

Radiographic diagnosis showed 226 foals with signs of osteochondrotic lesions. In 107 foals osteochondrotic lesions were exclusively diagnosed in the fetlock joints, 59 foals revealed osteochondrotic lesions exclusively in the hocks, 28 foals had osteochondrotic lesions exclusively in the stifles, and 32 foals exhibited osteochondrotic changes in varying joints. Fillies revealed a higher incidence of OC in the fetlock joints, whereas colts had a higher manifestation of OC in the hocks.

Mean birth weights of the foals were  $57.3 \pm 8.1$  kg (N = 166). The patterns of growth rate are shown in Table 1. A proper model to describe body weight development (0 - 250 day of life) seemed to be the Boltzmann function with the following equation:  $y = A2 + (A1 - A2)/(1 + \exp((x - x0) / dx))$ , where y = bodyweight [kg], x = age [days], A1 = -280.8, A2 = 348.5, x0 = 19.44, and dx = 116.2.

Sex, birth month, the development of body weight, and withers height were not statistically different between healthy foals and those foals who developed osteochondrotic lesions. However, osteochondrotic lesions in the fetlock joints were associated with a lower body weight, whereas changes in the hocks were linked with a significantly higher body weight in the fourth and fifth month of life when compared to healthy foals (Table 2).

*Table 1. Body weight [kg] in foals.*

| age [d]   | body weight [kg] |    |     |                  |    |     |
|-----------|------------------|----|-----|------------------|----|-----|
|           | OC neg.          |    |     | OC pos.          |    |     |
|           | mean             | SD | N   | mean             | SD | N   |
| 0 - 30    | 81 <sup>A</sup>  | 17 | 302 | 84 <sup>A</sup>  | 15 | 165 |
| 31 - 60   | 120 <sup>B</sup> | 19 | 361 | 122 <sup>B</sup> | 18 | 195 |
| 61 - 90   | 155 <sup>C</sup> | 20 | 376 | 155 <sup>C</sup> | 21 | 203 |
| 91 - 120  | 187 <sup>D</sup> | 22 | 370 | 186 <sup>D</sup> | 23 | 209 |
| 121 - 150 | 216 <sup>E</sup> | 23 | 353 | 214 <sup>E</sup> | 26 | 209 |
| 151 - 180 | 242 <sup>F</sup> | 26 | 296 | 236 <sup>F</sup> | 27 | 170 |
| 181 - 210 | 263 <sup>G</sup> | 27 | 89  | 256 <sup>G</sup> | 28 | 56  |

means lacking a common superscript differ (P<0.001) within a column

*Table 2. Body weight [kg] in foals with regard to the location of osteochondrotic lesions.*

| age [d]   | body weight [kg]  |    |     |                   |    |    |                  |    |    |                   |    |    |
|-----------|-------------------|----|-----|-------------------|----|----|------------------|----|----|-------------------|----|----|
|           | OC neg.           |    |     | OC fetlock joints |    |    | OC hock          |    |    | OC stifles        |    |    |
|           | mean              | SD | N   | mean              | SD | N  | mean             | SD | N  | mean              | SD | N  |
| 0 - 30    | 81 <sup>a</sup>   | 17 | 302 | 82 <sup>a</sup>   | 15 | 79 | 87 <sup>a</sup>  | 14 | 41 | 87 <sup>a</sup>   | 17 | 18 |
| 31 - 60   | 120 <sup>a</sup>  | 19 | 361 | 119 <sup>a</sup>  | 17 | 90 | 122 <sup>a</sup> | 21 | 48 | 124 <sup>a</sup>  | 19 | 22 |
| 61 - 90   | 155 <sup>ab</sup> | 20 | 376 | 150 <sup>a</sup>  | 20 | 93 | 160 <sup>b</sup> | 23 | 54 | 157 <sup>ab</sup> | 20 | 21 |
| 91 - 120  | 188 <sup>a</sup>  | 22 | 370 | 178 <sup>b</sup>  | 21 | 95 | 195 <sup>c</sup> | 25 | 58 | 190 <sup>ac</sup> | 19 | 21 |
| 121 - 150 | 216 <sup>a</sup>  | 23 | 353 | 206 <sup>b</sup>  | 23 | 98 | 223 <sup>c</sup> | 30 | 55 | 216 <sup>ac</sup> | 20 | 21 |
| 151 - 180 | 242 <sup>a</sup>  | 26 | 296 | 231 <sup>b</sup>  | 24 | 86 | 246 <sup>a</sup> | 34 | 43 | 235 <sup>ab</sup> | 25 | 14 |
| 181 - 210 | 263 <sup>a</sup>  | 27 | 89  | 244 <sup>b</sup>  | 25 | 24 | 269 <sup>a</sup> | 30 | 16 | 254 <sup>ab</sup> | 18 | 4  |

means lacking a common superscript differ (P<0.05) within a row

## Conclusions

The body weights used by the German recommendations (GEH 1994) to calculate nutrient requirements for foals seemed to be appropriate. The present study did not indicate significant differences in body weight and withers height between OC affected or OC unaffected



Warmblood foals. Differences in body weight were noticed with regard to the location of osteochondrotic lesions. These findings were confirmed by Jelan *et al.* (1996) in Thoroughbred horses (N = 798). It is assumed that there are different pathogenic factors for the development of osteochondrotic lesions in horses.

In consequence, a high body weight might be detrimental in those foals who were genetically predisposed with osteochondrotic lesions in the hock. However, in this Hanoverian foal population growth rates were quite moderate and body weight should not be overestimated as several etiological factors might play a role in osteochondrosis.

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## **Part B**

### **Energy and protein requirements**



# Energy and protein metabolism of normal growth

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## Abstract

Energy and protein intake are the primary factors influencing the growth rate of young horses. Normal growth includes both in utero and postnatal tissue deposition, including muscle, connective tissues, internal organs and bone. There appears to be a relationship between energy and protein needs of the growing horse that may serve to simplify the complicated involvement of both energy and protein for growth into a usable formula for providing accurate feeding programs. The growing horse requires energy for both maintenance and growth. The relationship between energy and protein needs for these two functions are different, so the requirements change as the animal matures. In addition to the energy:protein relationship there is also the animal's need for amino acids, the building blocks of protein. The amino acid content of dietary protein often varies considerably from the animal's requirements for efficient protein synthesis for building tissues. The variation in amino acid prececal absorption, utilisation and turnover make planning the diet for maximum efficiency important for both the animal's well being and economic efficiency.

*Keywords: horse, energy, protein, amino acids*

## Introduction

Growth and development of the young horse is dictated by genetics and the energy and protein intake of the animal. Growth includes the deposition of muscle, skeletal, and internal organ tissues. In most commercial farms, growth rate is controlled by the amount of energy and protein provided to the animal. The definition of normal growth varies with age, breed and purpose. The Thoroughbred that is destined to be sold in a yearling sale of prospective race horses must be grown out at its maximum potential in order to be competitive in the sales ring. That same animal produced for the hunter/jumper market and probably not destined to seat a rider until he is three years of age can be grown out more slowly. This variation in growth rate will influence our discussion and perhaps how the information there in is applied to various horse management systems. In most of our review, we will assume that maximum growth is desirable even though it may not always be necessary.

## Growth in utero

Fetal development proceeds quite slowly during the first 8 months of gestation. In the third trimester, about 60% of the development occurs and the increase in weight is almost linear (Figure 1) during this time (Meyer and Ahlswede, 1978; Platt, 1984). This means that for a 50 kg foal only about 0.08 kg of tissue is deposited daily during early gestation and that during late gestation this deposition rate increases to 0.33 kg/d or more. This fetal and supporting tissue deposition influences the nutrient needs of the mare and fetal development can probably be influenced at least in a modest way by the energy and protein intake of the mare during late gestation. At birth a typical Thoroughbred foal will weigh 52.6 kg and will be composed of 27.3% DM, 17.1% protein, 2.1% NFE, 2.6% fat and 5.6% ash. On a DM basis

the composition would be 62.6% protein, 7.7% NFE, 9.5% fat and 20.5% ash (Meyer and Ahlswede, 1978).

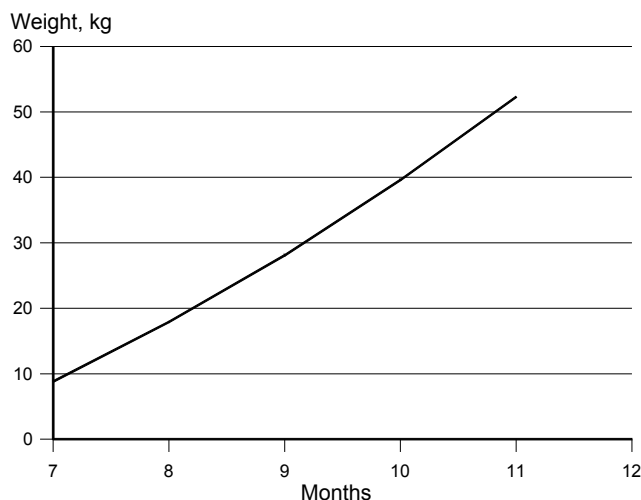


Figure 1. Fetal growth for Thoroughbred foals (Meyer and Ahlswede, 1978).

### Growth of the suckling

After parturition, growth of the suckling foal is dependent primarily on the milk production of the mare during early lactation and on available supplemental nutrients during late lactation. Thoroughbred and Quarter Horse mares (34) with a mean weight of 572.5 kg produced foals that weighed 52.0 kg at birth (9.1% of mares BW) and gained 1.38 kg/d for 112 d (Ott and Asquith, 1994). In a subsequent study 27 mares of similar size had foals that weighed 52.8 kg at birth and gained 1.26 kg/d for 112 days (Kavazis *et al.*, 2002). Thompson *et al.*, (1995) did not record birth weights but measured 112 day weights at 193.6 kg for colts and 194.5 for fillies. If we assume similar birth weights to those shown above the daily gains would be 1.26 and 1.27 kg/d for colts and fillies, respectively. As the foal gets older, he relies less on the mare's milk and more on supplemental feed. Coleman *et al.*, (1999) reported that from 67 to 120 day of age foals gained 1.06 kg/d with no creep feed and 1.20 and 1.14 kg /day when offered two creep feeds. Kavazis and Ott, (2003) reported on the growth of 141 Thoroughbred foals. Weight at birth was  $53.6 \pm 5.2$  kg and the weight from birth to 112 d was represented by the equation  $Y_1 = 1.28x + 57.82$  ( $r^2 = 0.94$ ). Where x = age in days.

### Growth of the weanling

Growth of the weanling will be influenced by genetic potential, growth rate during the suckling period, weaning stress and energy and protein intake of the animal. If the foal is not adequately supplemented during lactation, some compensatory growth would be expected. Growth rates of Thoroughbred and Quarter Horse weanlings 4 to 8 months of age at our station have been 0.69, 0.77, and 0.76 kg/d in three experiments (Ott and Kivipelto, 2002). Weight for 141 Thoroughbred foals from 113 to 540 d was represented by the equation  $Y_2 = 0.57x + 133.28$  ( $r^2 = 0.86$ ) (Kavazis and Ott, 2003).

## **Growth of the yearling**

Yearling growth rate is influenced by genetic potential, growth prior to the yearling period, energy and protein intake. If no compensatory growth is expected, growth of the yearling will be slower than the two earlier periods.

## **Growth of the two-year-old**

Under most circumstances the growth of the two-year-old Thoroughbred horses will be less than 0.5kg/d

## **Skeletal development**

Bone development in the growing horse is initiated *in utero* and continues until the animal is about 5 years of age, although most of the limb development is completed by 36 months of age. Bone consists of both organic and inorganic fractions. The organic fraction is built first and then mineralized to provide the support structure for the animal. Meyer and Ahlswede (1978) reported that bone of newborn foals contained 63.2% ash, 36.8% organic matter and 31.4% protein on a dry matter basis. However, Elshorafa *et al.* (1979) reported ash values in newborn foals to 8 mo of age to be 55.2% and did not reach 64% until 4 to 7 yrs of age followed by a gradual decline. Voges *et al.* (1990) recorded 61.8% ash in third metacarpals of foals < 6 mo. of age increasing to 69.6% at 61 - 120 mo. of age. These increases in bone ash are consistent with Lawrence *et al.* (1994), who found that bone mineral increased from birth to  $6.0 \pm 1.8$  yrs of age. The correlation between bone mineral content and breaking strength in their study was high ( $r^2 = 0.84$ ).

## **Energy metabolism**

Energy metabolism can be considered at the cellular level, the system level or the whole body level. For the purposes of this discussion we will consider the whole body energy needs for growth. However, we should recognize that there may be some system and cellular changes that control the body's needs. Energy metabolism must be divided into maintenance and growth contributors to the animal's total needs.

Maintenance energy requirements include the basal metabolic rate (BMR) of the animal, plus energy needs for normal movement and feed consumption. Although we have generally concluded that the maintenance needs are a function of body weight or metabolic body weight, the variation in voluntary activity may be a large factor in actual maintenance needs. For example, the activity of an animal grazing a large pasture will be considerably higher than that of an animal confined to a box stall with minimum turn out. The magnitude of this difference has not been clearly defined in the growing horse. It is also of interest to look at the effects of temperature on maintenance requirements. As temperature falls below the animals thermal neutral zone, energy expenditure to sustain body temperature will add to the total energy needs of the animal.

It is generally assumed that the growth requirements and the maintenance requirements are additive and as the animal matures, maintenance becomes a larger portion of the needs (Table 1). Note that for all but one example the maintenance requirement is greater than the growth requirement.

Table 1. Digestible energy requirements for maintenance and growth of selected horses<sup>1</sup>.

|                            | Maintenance<br>Mcal/d | Growth<br>Mcal/d | Total<br>Mcal/d |
|----------------------------|-----------------------|------------------|-----------------|
| Weanling, 215 kg           |                       |                  |                 |
| Moderate growth, 0.65 kg/d | 7.85 (52.3%)          | 7.15 (47.7%)     | 15.0            |
| Rapid growth, 0.85 kg/d    | 7.85 (45.6%)          | 9.35 (54.4%)     | 17.2            |
| Yearling, 325 kg           |                       |                  |                 |
| Moderate growth, 0.50 kg/d | 11.15 (59.0%)         | 7.75 (41.0%)     | 18.9            |
| Rapid growth, 0.65 kg/d    | 11.15 (52.3%)         | 10.15 (47.7%)    | 21.3            |
| Long yearling, 400 kg      |                       |                  |                 |
| Moderate growth, 0.35 kg/d | 13.4 (67.7%)          | 6.4 (32.3%)      | 19.8            |

<sup>1</sup>NRC 1989 Based on the equation  $DE = (1.4 + 0.03 BW) + (4.81 + 1.17x - 0.023X^2)ADG$  where X = age in months and BW and ADG are in kg.

There are three aspects of energy intake by the growing horse that are relevant to this discussion. First, energy is required for virtually all body functions and consequently influences the rate at which these functions occur. The NRC (1989) estimated the energy requirements of the growing horse using the equation:

$$DE_{(Mcal)} = (1.4 + 0.03 BW_{kg}) + (4.81 + 1.17x - 0.023X^2) ADG$$

Where x = age in months and ADG is in kg/d.

The maintenance component of this equation gives a requirement of a 325 kg animal at 11.15 Mcal DE/d. Cymbaluk *et al.* (1989) reported maintenance requirements for animals 6 to 12 months of age at 37.8 and 35.6 kcal DE/kg BW daily for limit fed and ad libitum fed animals. This would be 12.3 and 11.6 Mcal DE daily, slightly more than the equation calculates. However, their diets were 60% or more roughage which would likely reduce the metabolic efficiency when compared to higher concentrate diets. They found that DM intake was largely related to body weight but was also influenced by temperature. For animals 6 to 12 months of age they observed a 0.2% increase in feed intake for each 1 °C decrease in temperature below 0 °C.

Using the above equation, the energy requirement for growth above maintenance would be 11.0 to 15.5 Mcal/kg gain from 6 to 12 mo of age. These results were similar to some of our studies (Table 2). We found weanlings needed 8.76 to 10.28 Mcal/kg gain and yearlings needed 13.9 to 20.5 Mcal/kg gain. The efficiency of using the energy was influenced by age and apparently the amount of energy intake. At high intakes the animal apparently used more of the energy for fat deposition.

Limiting energy intake will reduce growth rate of young animals, including the rate at which bone is mineralized (Ott and Asquith, 1986; Thompson *et al.*, 1988). This relationship between energy intake and bone development dictates that as energy intakes are increased, the other nutrients required for bone development increase to insure adequate nutrient pool for optimizing both bone matrix deposition and bone mineralisation. Second, the high energy intake may have detrimental effects on skeletal development. High levels of energy stimulate growth, particularly fat accumulation, and seem to have a detrimental effect on skeletal quality or at least its resistance to stress. Tilburg and Ellis (2002) reported that Dutch Warmbloods with OC problems had higher growth rates than those without OC problems.



There was no information available on the energy intake of these animals. However, Vervuert *et al.*, (2003) did not detect any effect of growth rate of Hanovarian Warmblood foals on OC lesions. In their study, 226 foals exhibited lesions of the 629 animals studied. Third, energy sources may alter hormone levels causing skeletal problems. High starch intakes result in hyperglycemia and hyperinsulinaemia and altered concentrations of T<sub>3</sub> and T<sub>4</sub>. (Glade and Belling, 1986; Glade and Luba, 1987). Both of which are involved in chondrocyte development and maturation. A relationship between hyperglycemia and hyperinsulinaemia and OC has been reported (Ralston, 1996).

Table 2. Energy requirement for growth.

| Group, Exp. and age    | Av. Wt.<br>Kg | DE Intake<br>Mcal/d | Maint. DE <sup>1</sup><br>Mcal/d | Gain DE <sup>2</sup><br>Mcal/d | ADG<br>kg | DE/kg<br>gain |
|------------------------|---------------|---------------------|----------------------------------|--------------------------------|-----------|---------------|
| Weanlings <sup>3</sup> |               |                     |                                  |                                |           |               |
| Exp. 1 - 244 d         |               |                     |                                  |                                |           |               |
| A                      | 234           | 14.77               | 8.42                             | 6.35                           | 0.68      | 9.33          |
| B                      | 236           | 15.57               | 8.48                             | 7.09                           | 0.69      | 10.28         |
| Exp. 2 - 250 d         |               |                     |                                  |                                |           |               |
| A                      | 218           | 15.32               | 7.94                             | 7.38                           | 0.79      | 9.34          |
| B                      | 224           | 14.94               | 8.12                             | 6.82                           | 0.74      | 9.22          |
| Exp 3 - 268 d          |               |                     |                                  |                                |           |               |
| A                      | 257           | 16.49               | 9.11                             | 7.38                           | 0.72      | 10.25         |
| B                      | 265           | 16.36               | 9.35                             | 7.01                           | 1.80      | 8.76          |
| Yearlings <sup>4</sup> |               |                     |                                  |                                |           |               |
| Exp 4 - 409 d          |               |                     |                                  |                                |           |               |
| A                      | 384           | 26.48               | 12.92                            | 13.56                          | 0.66      | 20.5          |
| B                      | 384           | 27.48               | 12.92                            | 14.57                          | 0.71      | 20.5          |
| Exp. 5 - 386 d         |               |                     |                                  |                                |           |               |
| A                      | 362           | 19.60               | 12.26                            | 7.34                           | 0.49      | 15.0          |
| B                      | 369           | 23.57               | 12.47                            | 11.10                          | 0.59      | 18.8          |
| C                      | 372           | 23.63               | 12.56                            | 11.10                          | 0.62      | 17.9          |
| Exp 6 - 385 d          |               |                     |                                  |                                |           |               |
| A                      | 323           | 17.70               | 11.09                            | 6.61                           | 0.48      | 13.8          |
| B                      | 331           | 22.10               | 11.33                            | 10.80                          | 0.63      | 17.1          |
| C                      | 316           | 17.28               | 10.88                            | 6.40                           | 0.46      | 13.9          |

<sup>1</sup>Calculated as DE maint = 1.4 + 0.03BW

<sup>2</sup>Calculated as total DE intake minus DE maintenance.

<sup>3</sup>Ott and Kivipelto, 2002

<sup>4</sup>Ott and Asquith, 1986.

Fat has become recognized as a useful source of energy for horses (Bowman *et al.*, 1980; McCann *et al.*, 1987). Fatty acids offer an alternative to a portion of the carbohydrates for supporting the energy demands of the rapidly growing horse. Lawrence *et al.* (1989) reported that fat supplementation of diets for growing yearlings reduced blood glucose and insulin concentrations following meals. The high fat diet also lowered T<sub>4</sub> concentrations but did not affect the T<sub>3</sub> values. In a study with 30 weanling Quarter Horses, animals fed a 10% fat concentrate with the same calorie:nutrient ratio as a typical high carbohydrate concentrate grew faster (0.80 vs 0.74 kg/d) and were more efficient. There were no radiographic abnormalities detected in either group and epiphyseal closures and bone density was the same for both groups (Davison *et al.* 1991). In a similar study with yearlings, the addition of 5% fat

to a concentrate, adjusted to provide the same calorie:nutrient ratio, resulted in the same rate of gain and no adverse affect on skeletal development (Ott and Kivipelto, 1997). Fat supplementation reduced blood insulin concentrations but did not affect blood glucose, perhaps because the starch content of the concentrate was still quite high. Growing foals fed a high fat supplement and pasture had lower bone mineral content than foals fed a conventional high starch concentrate, suggesting that some highly available carbohydrate is essential for maximizing skeletal development (Hoffman *et al.*, 1999).

The type of fatty acids in the diet may influence bone mineralisation. Watkins *et al.* (1997) found that butter + corn oil (BC) had a positive effect on bone formation in chicks compared to soybean oil (SBO), margarine + corn oil (MAC) or Menhaden oil + corn oil (MEC). It appears that BC lowered bone arachidonic acid (20:4, n-6) but raised IGF-I content, moderated PGE<sub>2</sub> production and resulted in increased bone formation. Yong *et al.* (1999) reported that rats fed conjugated linoleic acid (CLA) had negative effects on bone PGE<sub>2</sub> resulting in lower mineral apposition and lower bone formation rates independent of the other source of fatty acids in the diet. Increased bone mineral density was also detected when (n-3) fatty acids in the diet were increased. This work implies that milk fat, a rich source of CLA, may have positive effects on bone mineralisation in young horses and older animals may benefit from (n-3) fatty acids.

## Protein metabolism

There are two components of interest in the protein metabolism of the growing horse. The maintenance component which is identified as the daily endogenous nitrogen loss and the protein deposition component or the amino acids required for tissue growth.

The endogenous fecal nitrogen loss includes the unabsorbed nitrogen from enzymes secreted into the digestive tract and the sloughing of cells from the gut wall. The basic value for metabolic fecal nitrogen (MFN) proposed by Maynard *et al.* (1979) of 2 mg/g DM intake may be too simple for most applications. Glade (1984) reported MFN values of 3.7 to 5.6 mg /g DM intake of concentrate and concentrate/forage combinations. Gibbs *et al.* (1988) provided information on the nitrogen digestion of three hays. Their results provided a MFN of 9.1 mg/g DM intake. Farley *et al.* (1995) reported 2.6 mg MFN/g DM intake on diets with different protein concentrations and Almeida *et al.* (1999) reported 5.1 mg MFN/g DM on 50:50 concentrate:forage diets. With the exception of the work by Almeida *et al.* (1999) most of the measurements of endogenous nitrogen has been made on mature animals. It is not clear whether the growing horse has the same endogenous loss as the mature animal. Endogenous fecal losses are apparently influenced by total feed intake and fiber content of the diet.

Urinary endogenous nitrogen loss is a function of muscle tissue turnover, which may be a major factor in young growing horses, and the deamination of amino acids so the carbon skeletons can be used as energy or other metabolic products. Urinary metabolic nitrogen has been reported as 153.0 mg/kg<sup>0.75</sup> (Hintz and Schryver, 1972), 126 mg/kg<sup>0.75</sup> (Glade, 1984), 135 mg/kg<sup>0.75</sup> (Meyer *et al.*, 1985), and 339 mg/kg<sup>0.75</sup> (Almeida *et al.*, 1999). In addition to endogenous losses, the amount of protein intake above the requirement will influence urinary nitrogen losses (Table 3).

Nitrogen digestion in the growing horse is reported as both apparent and true digestion. Apparent digestibility ( $\text{Apparent dig.} = \frac{\text{intake} - \text{excretion}}{\text{intake}}$ ) is probably not very useful

since it does not reflect the actual availability of amino acids to the animal. By subtracting endogenous nitrogen from the excreted nitrogen a correction is made for the nitrogen lost via the feces from the animal. Under these circumstances the true digestibility of the nitrogen will usually be 85 to 95%. However, this also does not reflect the actual value of the protein to the animal since what we really need to know is how much of the protein is digested in the foregut (small intestine). That which escapes foregut digestion and absorption is broken down almost totally by the microbial population of the cecum and colon, being utilized by the organisms as either a protein source or an energy source. Consequently, only a small amount of the nitrogen absorbed in the hind gut is in the form of usable amino acids. Foregut digestibility is likely influenced by diet form and composition. Gibbs *et al.* (1988) reported that only a small portion of the nitrogen from hay was digested in the foregut (37%) but Farley *et al.* (1995) provided evidence that 72% of the nitrogen from soybean meal was digested in the foregut. This suggests that we must consider not only the type of diet but also the source of nitrogen when predicting the availability of nitrogen to the animal. There is also some indication that the foregut has a limit as to how much nitrogen it can handle in a meal. Farley *et al.* (1995) suggested that 125 to 150 mg N/kg BW per meal is the upper limit in a mature animal. The limit in the growing horse has apparently not been established.

The utilisation efficiency of absorbed nitrogen is also of interest. The source of amino acids and probably age influences the efficiency of utilisation. Hintz and Schryver (1972) reported an efficiency of 45.5%, Meyer *et al.* (1985) reported 74.0% and Almeida *et al.* (1999) reported 56.6%.

Table 3. Nitrogen intake and excretion of yearling horses consuming various protein concentrations<sup>1,2</sup>.

|                      | CP in diet, % |       |       |       |       | Y =             |
|----------------------|---------------|-------|-------|-------|-------|-----------------|
|                      | 7.45          | 10.26 | 13.23 | 16.04 | 18.51 |                 |
| Nitrogen intake, g/d | 81.6          | 125.9 | 162.3 | 166.7 | 234.8 | - 9.49+12.43 CP |
| Nitrogen fecal, g/d  | 32.2          | 30.8  | 34.3  | 38.1  | 35.8  | 34.7            |
| Nitrogen urine, g/d  | 12.0          | 38.5  | 63.5  | 58.0  | 132.7 | -63.39+9.43 CP  |
| Nitrogen absor, g/d  | 49.4          | 95.1  | 128.0 | 128.6 | 198.9 | -36.90+11.90 CP |
| Nitrogen ret., g/d   | 37.4          | 56.6  | 64.5  | 70.6  | 66.2  | 60.6            |
| Nitrogen ret., %abs  | 75.7          | 61.3  | 50.6  | 51.0  | 34.8  | 52.7            |

<sup>1</sup> Almeida *et al.*, 1999

<sup>2</sup> 330 kg BW

Tissue deposition is dependent upon the availability of appropriate concentrations of amino acids. The amino acid content of horse muscle tissue has not been well documented and may represent a key to the optimum amino acid needs of the growing horse. However, digestion and absorption of the amino acids and turn over rate is also important. About 75% of the dietary amino acids are absorbed in the foregut (small intestine) but there is little evidence available of differences in the absorption of individual amino acids by the horse.

The importance of protein quality, that is, the amino acid content of the protein and the availability of those amino acids to the growing horse, has been demonstrated by a number of investigators (Hintz *et al.*, 1971; Borton *et al.*, 1973; Potter and Hutchton, 1975; Ott *et al.*, 1979). In all of these experiments, lysine seemed to be the first limiting amino acid. This was verified by Ott *et al.* (1981). Their results suggested that the lysine requirement of the

yearling was 1.9 g/Mcal DE. Saastamoinen and Koskinen (1993) provided evidence that protein quality has a significant effect on growth response in weanling horses. They compared a milk protein based diet with a barley protein based diet. The milk protein diet resulted in increased weight gain, longissimus dorsi muscle growth, cannon bone circumference, serum amino acids, hemoglobin and hematocrit. They calculated that the weanling needs 31 g lysine/d or 0.46 g lysine/MJ ME (2.2 g/Mcal DE) The second limiting amino acid in concentrate/grass forage diets (Graham *et al.*, 1994) is apparently threonine. This relationship was supported by Staniar *et al.* (1999) who reported that growing horses fed a 9% CP supplement fortified with 0.6% lysine and 0.4% threonine supported the same growth rate as a 14% protein supplement.

However, the amounts of these amino acids required in the diet is influenced by the efficiency with which they are absorbed from the small intestine. Almeida *et al.* (1999) fed diets providing 50:50 concentrate:hay to yearling horses. The concentrates provided increasing amounts of protein from 7.45% to 18.51%. The supplemental protein was provided by soybean meal. Foregut digestibility of the five diets is shown in Table 4.

Table 4. Influence of CP intake on prececal amino acid digestibility (%).

| CP, %       | Diet      |             |              |             |            |                  |
|-------------|-----------|-------------|--------------|-------------|------------|------------------|
|             | I<br>7.45 | II<br>10.26 | III<br>13.23 | IV<br>16.04 | V<br>18.51 |                  |
| Lysine      | 51.7      | 62.1        | 73.1         | 62.8        | 69.3       | Y=64.3           |
| Threonine   | 31.2      | 49.4        | 51.0         | 51.2        | 62.4       | Y=21.875+54.265X |
| Methionine  | 72.0      | 73.1        | 69.8         | 70.7        | 77.2       | Y=73.3           |
| Valine      | 47.0      | 55.2        | 61.4         | 52.0        | 62.4       | Y=49.3           |
| Leucine     | 72.4      | 72.4        | 73.9         | 67.9        | 74.3       | Y=72.1           |
| isoleucine  | 92.3      | 90.2        | 88.8         | 84.5        | 86.3       | Y=88.2           |
| Histidine   | 65.2      | 72.3        | 76.1         | 75.7        | 81.4       | Y=60.625+44.783X |
| Phenyl      | 91.9      | 69.7        | 88.5         | 84.2        | 87.8       | Y=84.7           |
| Arginine    | 67.3      | 79.9        | 82.7         | 83.4        | 85.7       | Y=67.014+16.175X |
| Tyrosine    | 84.6      | 83.7        | 81.6         | 81.8        | 85.7       | Y=83.3           |
| Cystine     | 39.5      | 45.4        | 56.1         | 58.8        | 71.9       | Y=24.167+19.298X |
| Aspartic a. | 36.0      | 59.4        | 60.6         | 51.7        | 61.2       | Y=54.1           |
| Glutamic a  | 56.8      | 43.0        | 55.9         | 59.1        | 52.2       | Y=55.6           |
| Alanine     | 47.0      | 44.4        | 46.4         | 51.0        | 44.0       | Y=46.8           |
| Glycine     | 36.6      | 48.6        | 50.6         | 45.9        | 52.8       | Y=47.1           |
| Serine      | 54.1      | 66.6        | 67.9         | 67.6        | 77.3       | Y=48.576+28.579X |

X=g amino acid in diet/kg DM

Almeida *et al.*, 1999.

Meeting the dietary protein requirement is actually dependent on not only the protein concentration in the diet but also feed intake and the amino acid content of the diet. Orton *et al.* (1985) demonstrated that yearlings fed an 8% protein diet could sustain normal growth (0.58 kg/d) when feed intake was stimulated by forced exercise. At high protein intakes, exercise was of no advantage. Protein intake did not influence bone development in this study. The amount of protein necessary for both growth and skeletal development in yearling horses seems to be about 40 g/Mcal DE (Ott and Asquith, 1986) which is less than NRC (1989) recommendations but is dependent upon adequate lysine intake.

It is important to look at protein requirements from two perspectives. The daily requirement and the dietary concentration. The daily protein requirement of the growing horse is influenced by the animal's daily maintenance requirement, or endogenous nitrogen loss, plus the rate of muscle tissue deposition. In addition, there appears to be two factors that influence the protein utilisation by the growing horse, the energy intake of the animal and the amino acid digestion and availability via the small intestine. If energy intake is inadequate, the animal will not respond to the protein and if the protein is inadequate, the animal will not respond to the energy. The NRC (1989) recommended 50 g CP/Mcal DE for weanlings, 45 g CP/Mcal DE for yearlings, and 42.5 g CP/Mcal DE for two-year-olds. They also recommended 2.1 g lysine/Mcal DE for weanlings, 1.9 g lysine/Mcal DE for yearlings and 1.7 g lysine/Mcal DE for two-year-olds. More recent data suggests that some adjustments may be appropriate.

Research with other species suggest that we should consider the following in developing recommendations on the amino acid requirements of the growing horse: 1) The true ileal digestibility of amino acids varies with the type of diet (Nyachoti *et al.*, (1997), 2) True ileal digestibility of amino acids also varies with the source of those amino acids (Nyachoti *et al.*, 1997), 3) The concept of ideal protein (Wang and Fuller, 1989) may make it unnecessary to establish the requirements of all amino acids once the requirement for the limiting amino acids are established (Tuitoek *et al.*, 1997). 4) The ideal protein may change as the animal matures (Hahn *et al.*, 1995).

## Conclusions

Energy and protein metabolism in the growing horse are closely related. It would therefore seem appropriate to express the protein and amino acid needs of the animal in terms of the energy needed to sustain the desired rate of growth. This relationship probably applies to other nutrients as well. By optimizing the energy:nutrient relationships we should be able to maximize growth and minimize the effect of nutrition on developmental abnormalities.

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# Evaluation of energy and protein requirements and recommended allowances in growing horses

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## Abstract

Original feeding standards /systems were proposed in the eighties in the USA (1978-1989) and in France (1984-1990). Several feeding standards were derived in Germany (GEH, 1994), in The Netherlands (CVB, 1996) and in Scandinavia (SCAND, 2002).

In all the feeding standards/systems, total energy requirements are broken down in maintenance and growth requirements. Maintenance requirements are estimated from body weight (BW) (NRC) or  $BW^{0.75}$  (all the others). Growth requirements are estimated from the variation of age (NRC-SCAND) or from the variation in the body composition drawn from allometry of body tissue (INRA) or estimated using relationship between body composition and the relative proportion of BW to adult weight at different ages (GEH-CVB). Growth requirements are calculated using the efficiency rates of energy for growth (GEH-CVB). The nutritional models performed by NRC and INRA were based or/and validated with feeding experiments.

In all the feeding standards/systems, total nitrogen requirements are broken down in maintenance and growth requirements for GEH, INRA, CVB and SCAND, whereas NRC is using a N/E ratio appropriate to the different classes of age. Maintenance requirements are estimated from  $BW^{0.75}$  for all the others. Growth requirements are predicted from the variation in chemical composition of body drawn from the allometry of body tissues (INRA) or estimated using a relationship between body composition and the relative proportion of BW to adult BW at different ages (GEH-CVB-SCAND). Growth requirements are calculated using efficiency rates of protein for growth (GEH-CVB-SCAND). The nitrogen requirements are based on and validated with feeding experiments for NRC and INRA.

Variations of energy and nitrogen requirements with age, BW and daily gain are discussed and compared across the different standards/systems. Recommended nutrients allowances in the different systems are presented and discussed across the different proposals. The main factors which are limiting the accuracy of the requirements and recommended allowances are discussed and some proposals are suggested to overcome them within the EU context.

*Keywords: Equine, growth, development, requirements, allowances, energy, protein.*

## Introduction

Horse production in Europe involves various types of equines which differ widely according to breed, management, growth rate, feeding system and age at breaking in, first training, dressage and competing as far as race, sport and leisure horses are concerned. Generally race horses are in training at 18 months to start racing at 2 years of age whereas sport horses are ridden in at 3 year olds then submitted to dressage to be ready for competing at 4 years of age for jumping or some time later for three days event, endurance and more advanced dressage. For leisure horses, first age of riding might be quite variable. As a result the growing period

of horses can account for 25 to 50% of the productive life according to disciplines. However, body weight and size gains during the first postnatal year can represent 50 to 70% of adult weight and body size.

Most European horse breeds, have been more or less specialised for race, sport or leisure along the time. As a result most young sports horses come from early maturing breeds. Contrary to other farm animals and coldblooded horses, sports horses (also defined as warmblood, standardbred or thoroughbred) are bred to be used until a late age (15-20 years). The composition of the body and the body gain during growth of these different breeds and types of animals can vary from one type to another, for a given live weight and live weight gain and this may lead to a significant diversity of nutritional requirements.

Requirements for equines were published in Europe by Kellener and Fingerling (1924), Hanson (1938), Axelsson (1943), Popov (1946) and Jespersen (1949), and in USA by Morrisson (1932). They were expressed in feed evaluation systems designed in Ruminants: TDN, ME, Fu for energy and DCP, DTCP for nitrogen. In the eighties new feeding standards were performed in and for equines in the USA (NRC 1978) and in France (INRA 1984) due to increase in the knowledge in Nutrition and in the growth of equines (see the two books both entitled “The Horse” published in USA and in France in 1990 and in 1984 respectively). By the nineties, these feeding standards/systems have been improved in the USA by NRC (1989) and in France by INRA (1990) and developed in other different European countries after some local adaptation due to new data obtained in the growth and in feeding of the young horses: Germany (GEH 1994), The Netherlands (CVB 1996) and the Scandinavian countries (Austbø 1996-2004), Italy (1993 and 2002), Spain (1994), Poland (1998) and Romania (1997) took on INRA systems without adaptation.

The aims of this publication are to determine:

- what are the current energy and protein requirements and allowances for growth in horses in Europe and in the USA;
- what is their basis and how do they compare;
- and to discuss the future possibilities for improving current energy and protein requirements and allowances for growing horses within the EU context.

## **Methods for evaluating nutrients requirements and recommended allowances**

### **Definitions**

Requirements and recommended allowances have to be clearly distinguished. The requirements stand for estimated or measured physiological expenditure of energy and protein in horses during different physiological situations. Allowances stand for the amount of nutrients, which are provided by the appropriate ration to meet the requirement of horses, and thus includes estimation of energy and protein provided by feed. Recommended allowances stand for the amount of nutrients provided by the ration optimised for meeting the goal of production and utilisation of horses – these link the breeders’ goal, the physiological requirements of the horse and feed allowances. It means that the amount of nutrients required for growth of the same horse, can be lower, equal or higher than the nutritional requirements depending on husbandry or/and economical aims.

## Methods for determination

Requirements are basically determined by factorial method. For maintenance, requirements are determined using balance experiments and then appropriate nutrients conversion rates for calculation. For growth, requirements are determined using growth curve, variation in body composition, based on average daily gain (ADG) and then nutrients conversion rates if these are known. Allowances are determined by global method e.g. feeding experiments carried out with a high number of animals during long period corresponding to husbandry system to include effect of individual animal variation, environmental factors as far as animals are expected to be in good health and well managed. In these experiments energy and nitrogen intakes are related to the true performances.

## Evaluation of energy requirements and allowances

### Calculation of energy requirements for growth in different countries: data base – methodology – requirements

In USA according to NRC 1989 (National Research Council) energy requirements for maintenance and growth are calculated by using the following equation:

$$DE = (\text{Maintenance}) + (4.81 + 1.17 \text{ Age} - 0.023 \text{ Age}^2) \text{ ADG} \quad R^2 = 0.99 \quad SE = 0.50$$

where: DE = Digestible Energy (Mcal/day)

BW = Body Weight (kg)

ADG = Average Daily Gain (kg)

Age = in months

The equation was calculated from experimental data (Householder *et al.*, 1977; Milligan *et al.*, 1985; Ott and Asquith, 1986; Schryver *et al.*, 1987; Scott *et al.*, 1987). The equations for maintenance requirements, defined as the amount of DE required for zero body weight change plus normal activity of non working horses) are given below. Requirements of horses weighing 600 kg or less are predicted from the equation Pagan and Hintz (1986a) and for more than 600 kg adapted from Pagan and Hintz (1986b) and Potter *et al.* (1987).

#### Maintenance

< 600 kg: DE (Mcal/day) =  $1.4 + 0.03 \text{ BW}$

> 600 kg: DE (Mcal/day) =  $1.82 + 0.0383 \text{ BW} - 0.000015 \text{ BW}^2$

In order to compare the different systems a common denominator must be found, and thus the proportional increased requirement in energy per kg of weight gain from 6 months, 1 year, 18 months and 2 years of age were chosen assuming a random 1 kg ADG throughout this period.

From point of weaning at 6 months to the one year old the DE (Mcal) requirements per kilogram of gain (day) increase by 40 p100. From the yearling to the two year old DE (Mcal) requirements per kilogram of gain (day) increase with the age of the young horse by a further 26 p100. This means that at two years the young horse requires 78 p100 more energy to gain an extra kilo of BW compared to the 6 months old (Table 1).

Table 1. Variation with age of energy requirements per kg daily gain (NRC, 1989).

| Age                   | BW kg | DE Requirements (Mcal)<br>for growth per kg of gain | p100 |
|-----------------------|-------|-----------------------------------------------------|------|
| Weanling, 6 months    | 215   | 11.0                                                | 100  |
| Yearling              | 325   | 15.5                                                | 141  |
| Long earling, 18 mths | 400   | 18.4                                                | 167  |
| 2 years old           | 450   | 19.6                                                | 178  |

Variation in total energy requirements due to variation in maintenance or/and growth requirements in young horses of 500kg adult BW must be studied by comparing step by step: the influence of BW(250kg vs 350kg) at the same high ADG (1kg/d)appropriate to these BW; then the influence of ADG (1.0Kg vs 0.5 kg/d) at the same BW(300kg). The comparison should be extended again step by step to determine the influence of heavier BW( 400kg vs 450kg) at the same high ADG: 0.5 kg/d; then the influence of ADG (0.5kg vs 0.2kg/d) at the same BW:425 kg.

For 1kg of daily gain total energy requirements is 32 p100 higher in yearling of 350 kg BW than in weanling of 250kg BW. Maintenance and growth requirements increase by 34 and 31p100 respectively. Comparing 1kg versus 0.5 kg daily gain in yearling of 300 kg BW, total requirements rise by 41 p100 whereas growth requirements increase by 200 p100.

For 0.5kg of daily gain total energy requirements are 23p100 higher in 2 years old than in yearling of 450kg and 400 kg respectively. Maintenance and growth requirements increase by +11 and +7p100 respectively. At 425kg BW total requirements increase by +32p100 as far as daily gain rise from 0.2 to 0.5 kg for growth requirements increase by 246 p100.

In Germany according to GEH 1994 (Gesellschaft für Ernährungsphysiologie) energy requirements for maintenance and growth are calculated by using the body weight determined by different workers (Crampton, 1923; Hesse, 1957; Flade, 1957; Cunningham and Fowler, 1961; Olofsson and Thomke, 1963; Jordan and Myers, 1972; Dusek, 1976; Reed and Dunn, 1977; Hintz *et al.*, 1979; Saastamoinen, 1990), derived daily gain, variation of the composition of the daily gain derived from the NRC (1978) equations and efficiency rate of energy conversion (assumed to be 60% for DE of feeds for growth drawn from other animal species Menke and Huss 1980).The system adds a 43 – 75p100 increase to the adult maintenance requirements for growing horses as given below:

|                                      |   |                    |
|--------------------------------------|---|--------------------|
| Maintenance DE (adults) (MJ/d)       | = | $0.6 * BW^{0.75}$  |
| Maintenance DE (to 6 mths) (MJ/d)    | = | $1.06 * BW^{0.75}$ |
| Maintenance DE (to 12 mths) (MJ/d)   | = | $0.84 * BW^{0.75}$ |
| Maintenance DE (to 18 mths) (MJ/d)   | = | $0.76 * BW^{0.75}$ |
| Maintenance DE (from 18 mths) (MJ/d) | = | $1.72 * BW^{0.75}$ |

Maintenance requirements included 20% additional energy for young horses managed with normal exercise.

$$DE = (\text{Maintenance}) + (\text{ADG} \times \% \text{ lipids in BW} / \text{Energy efficiency})$$

where DE (MJ/day) = Digestible Energy  
 ADG = average Daily Gain (kg)  
 $\% \text{lipids/BW} = 0.1388p + 1.111$  (from NRC, 1978)  
 BW in p100 of adult BW  
 Energy efficiency = 0.60  
 BW= Body Weight (kg)

DE requirements per kilogram of gain (Mcal/day) calculated at different ages, increases with age of the young horse from 19 to 47 p100 in the yearling and 2 year olds as far as weanling is to be the reference (Table 2).

*Table 2. Variation with age of energy requirements per kg daily gain (GEH, 1994). \**

| Age                   | BW kg | DE Requirements (Mcal)<br>for growth per kg of gain | p100 |
|-----------------------|-------|-----------------------------------------------------|------|
| Weanling, 6 months    | 225   | 3.6                                                 | 100  |
| Yearling              | 315   | 4.3                                                 | 119  |
| Long earling, 18 mths | 380   | 4.9                                                 | 136  |
| 2 years old           | 425   | 5.3                                                 | 147  |

\* converted to Mcal DE

For 1 kg daily gain, total requirements increase by 20p100 when body weight rises from 250 to 350 kg in the yearling versus weanling. The increase deals with maintenance and growth requirements: +16p100 and +35p100 respectively. But comparing 1kg versus 0.5kg daily gain in yearling of 300 kg BW, total requirements increase by 11 p100 as growth requirements rise by 200p 100. For 1kg daily gain total requirements are 10p100 higher in 2 years old than in yearling of 450kg and 400kg respectively. Maintenance and growth requirements are both concerned in this increase: +9p100 and +16p100 respectively. At 425 Kg BW total requirements increase by + 9p100 as far daily gain rise from 0.2 to 0.5kg for growth requirements increase by + 251p100.

In France according to INRA (Institut National Recherche Agronomique) 1984-1990 energy requirements have been evaluated from long term feeding trials involving young heavy horses (Agabriel *et al.*, 1984) and young warmblood horses: Anglo Arabian, Selle, Français (Bigot *et al.*, 1987; Trillaud-Geyl *et al.*, 1992; Micol and Martin-Rosset, 1995) in which energy intake, weight and weight gain of young horses were determined for three reasons ( Martin-Rosset *et al.*, 1994):

1. The energy requirement for maintenance per kg BW<sup>0.75</sup> (metabolic body weight) varies with breed and growth rate as is well known in other animals (cattle, pigs).The variations in maintenance requirements due to breed are known only in adults (see reviews of Olsson and Ruudvere, 1955; Vermorel *et al.*, 1984), and the proportion of maintenance requirements in total energy requirements of growing horses ranges between 50 to 90 p.100 according to age and daily gain (Martin-Rosset *et al.*,1994);
2. The amount of fixed energy per kg weight gain computed from the chemical composition of different tissues was determined only in heavy breeds ( Martin-Rosset *et al.*, 1983b);
3. The efficiency of metabolisable energy (ME) utilisation for growth is unknown yet. Energy allowances were computed according to BW and daily gain of young horses using a relationship performed from the results of feeding trials carried out at INRA according to the general following model:

$$NE = a + b.G^{1.4}$$

where: NE= Net Energy (UFC/kgBW<sup>0.75</sup>/d)

a = coefficient for maintenance requirements

b.G<sup>1.4</sup> = calorific value of daily gain

G = daily gain(Kg/d)

1.4 = allometry coefficient of adipose tissue in total body weight

This general model was validated from a physiological and nutritional point of view. Drawn from previous work conducted at INRA in heavy breeds between 0 to 3 years of age (Martin-Rosset *et al.*, 1983 b), muscles proportion in the gain is nearly constant as far as allometry coefficient (b)e.g. relative growth to empty BW is b =1.1, whereas skeleton proportion decreases proportionally because b= 0.7, but adipose tissue proportion rises tremendously b = 2.1. As a result anatomical composition changes during the growth: the higher the body weight is of the growing horse, the higher is the proportion of adipose tissue in the daily gain e.g. lipids content; thus calorific value of the gain rises. Average chemical composition (lipids, proteins, and water content of body mass (empty body weight) were determined from birth to 30 months of age from slaughtered and anatomically dissected animals (Martin-Rosset *et al.*, 1983b). It was stated that total lipids content is linked to body mass (empty body weight = EBW) accordingly to the following equation: Lipids = 0.03 EBW<sup>1.6</sup>. This relationship is very close to that observed in beef cattle (INRA, 1978) but water content of fat free body mass in the foal at birth and in the adult horse decreases from 74.6 % to 70.7 % respectively (Robb *et al.*, 1972; Meyer *et al.*, 1976) as in cattle (Robelin et Geay, 1978). Thus the following relationship was established:

$$WM = 0.832 FFM^{0.974}$$

WM= Water Mass in the body

FFM= Free Fat Mass (Empty body mass-lipids)

Proteins content of dry fat free body mass averages 78 % from Robb *et al.* (1972); Schryver *et al.*, (1974) and NRC (1978).

Using all these previous hypothesis, net requirements for growth were evaluated from lipids, proteins and water content of body mass. Variations of chemical composition of daily gain was modelled (Figure 1).

200 g, 115 g and 620 g of proteins, lipids and water are fixed respectively in 1000 g Empty Body Weight (EBW) gain at 300 kg EBW ; which accounts for 2.2 Mcal/kg EBW gain as far as calorific value is 5.38 kcal/g and 9.31 kcal/g for proteins and lipids respectively according to Robb *et al.* (1972). At 600 kg EBW, 200 g, 180 g and 570 g of protein, lipids and water content would be fixed respectively, which accounts for 2.7 Mcal/kg EBW gain. At the same BW=300kg, maintenance requirements reach 9.46 Mcal, but the cost of 1kg body gain increases with the level of growth:+ 1.76Mcal when growth requirements calculated for 0.5kg ADG(1.1 Mcal) are compared with the requirements for 1kg ADG:2.86 Mcal.

But chemical composition of EBW gain is affected too by diet composition e.g. nutrients concentration as it was determined in 125 horses slaughtered at 12 and 18 months of age and at the end of feeding experiments conducted at INRA (Table 3). Average chemical composition/kg of EBW reached 208 g protein and 130 g lipids and 2 320 kcal for young horse weighting lower than 400 kg EBW as provided out Figure 1. In addition lipids content increased with average daily gain (Figure 2).

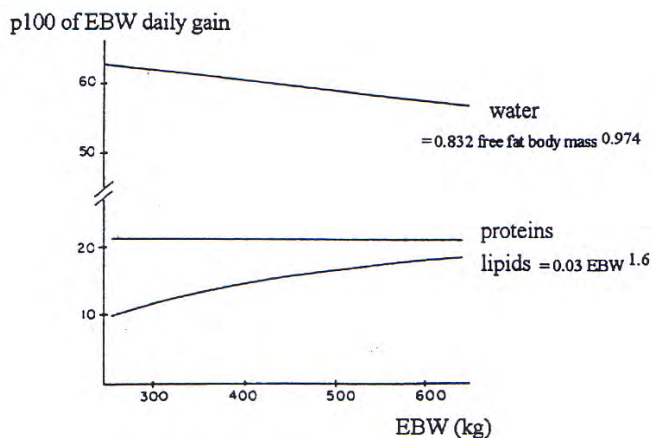


Figure 1. Variation of chemical composition during daily gain for growing horses (Source: Agabriel et al., 1984).

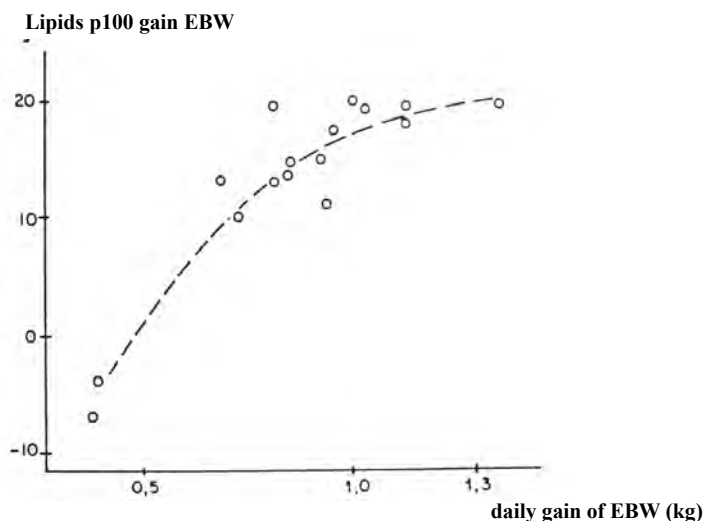


Figure 2. Lipid proportion of weight gained in relation to average daily gain (kg) (Source: INRA, 1984).

By example it rises from 0 to 20 p.100 per kg EBW gain and energy fixed from 830 to 2 060 kcal where EBW daily gain increases from 600 to 1300 g.

Using all this new knowledge two distinct groups of equations were developed for heavy and light breeds from feeding experiments data conducted in heavy and light breeds (Table 3) using the general model before simplification:

$$NE \text{ (UFC/d)} = aBW^{.75} + bBW^{.75} G^{1.4}$$

Where NE intake is broken down in two parts:

- $aW^{0.75}$  is proportional to metabolic weight (0.75) as for maintenance

- $\text{BW}^{0.75} \text{G}^{1.4}$  is the calorific value of gain (G) which is linked to intensity of G and body weight (BW). BW is affected by 0.75 exponent similarly to that for maintenance too, as a result it allows to compare energy intake ( $\text{UFC/kg BW}^{0.75}$ ) at different body weight and the exponent of 1.4 for G was designed.

This primary equation was further simplified to calculate the requirements as following:

$$\text{NE (UFC/kgW}^{0.75} \text{/day)} = a + b\text{G}^{1.4}$$

where: NE = Net Energy expressed in UFC (= Horse Feed Unit)

BW<sup>0.75</sup> = Metabolic body weight

G = Gain (kg/d)

1.4 = allometry of fat

a = maintenance requirement

$b\text{G}^{1.4}$  = calorific value of gain

Energy requirements for maintenance expressed per kg BW<sup>0.75</sup> are higher (+ 21 p.100 at 12 months by example) in light breeds than in heavy breeds, and they decrease with age in each breed (Table 3).

Energy requirements for growth are different according to body weight, daily gain and calorific value of gain whatever the breed (Table 3).

*Table 3. Values of the coefficients a and b for calculating NE requirements (UFC/kgW<sup>0.75</sup>/day) for light and heavy horsebreeds (INRA, 1990).*

| Age (months) | Light breeds       |                                     | Heavy breeds       |                                     |
|--------------|--------------------|-------------------------------------|--------------------|-------------------------------------|
|              | BW <sup>0.75</sup> | BW <sup>0.75</sup> G <sup>1.4</sup> | BW <sup>0.75</sup> | BW <sup>0.75</sup> G <sup>1.4</sup> |
| 6 - 12       | 0.0602             | 0.0183                              | 0.0476             | 0.0254                              |
| 18 - 24      | 0.0594             | 0.0252                              | 0.0485             | 0.0350                              |
| 30 - 36      | 0.0594             | 0.0252                              |                    |                                     |

In light breeds, for 1kg of daily gain, energy requirements increase by 35 to 137 p100 in the yearling and in the 2 years old as far as the weanling is to be the reference (Table 4).

*Table 4. variation with age of energy requirements per kg daily gain in light breeds (INRA, 1990).*

| Age                    | BW (kg) | NE (UFC) requirements for 1 kg ADG | Total NE (Mcal) requirements for 1 kg ADG | Increase p100 from weanling value |
|------------------------|---------|------------------------------------|-------------------------------------------|-----------------------------------|
| Weanling, 6 months     | 220     | 1.04                               | 2.4                                       | 100                               |
| Yearling               | 325     | 1.40                               | 3.1                                       | 135                               |
| Long yearling, 18 mths | 400     | 2.25                               | 5.0                                       | 216                               |
| 2 years old            | 450     | 2.46                               | 5.4                                       | 237                               |

For a constant daily gain, the intake of UFC increases less and less: CB=BA (0.3 kg ADG) then CB>BA (0.7 kg ADG) then CB>>BA (0.9 kg ADG) because in the designed model,



energy intake is proportional to  $BW^{0.75}$  ( $0.75 < 1$ ) and maintenance becomes increasingly important as the foal gets older (Figure 3).

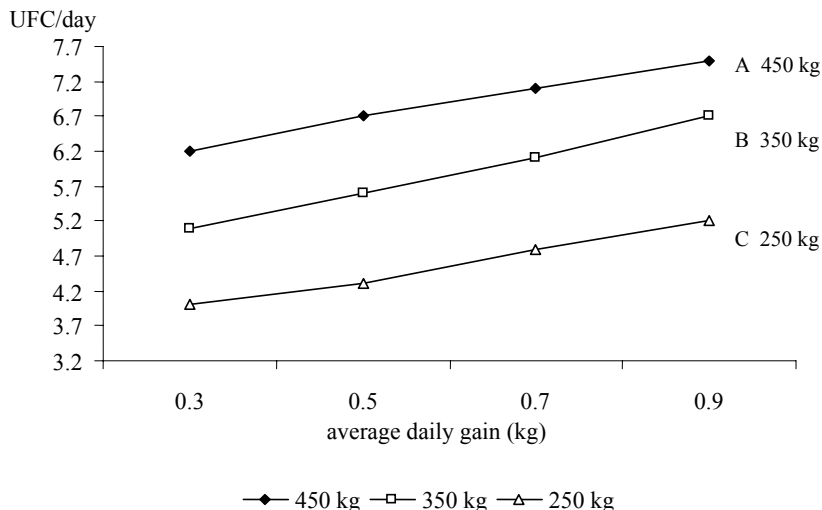


Figure 3. Variation in UFC intake with BW increase and changes in ADG for three final adult bodyweights (Source: INRA, 1990).

For a constant body weight the ratio UFC/kg of daily gain rises as far as daily gain increases but more and more due the exponent 1.4 of G (Figure 3). For 1 kg of daily gain, total requirements are 31p100 higher in yearling of 350 kg BW than in weanling of 250 kg BW (Table 8). The increase deals with the maintenance and growth requirements: + 29 and +36 p100 respectively. Comparing 1kg versus 0.5 kg daily gain in the yearling of 300 kg, total requirements increase by 11p100 whereas growth requirements rise by 186 p100. Variation of energy requirements for growth with daily gain is extensively described by the regression between energy intake and daily gain (Figure 4). Individual variability is rather high (RSD = 0.0084) as far as horses have not been selected for growth capacity.

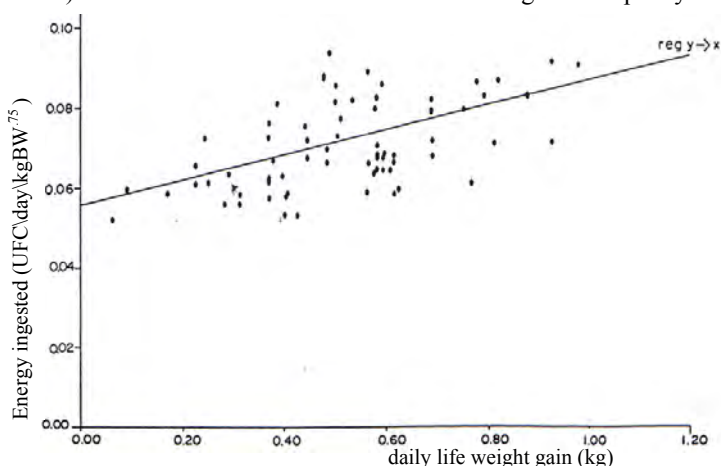


Figure 4. Relationship between energy intake and daily gain for light breed horses (Source: INRA, 1984).

For 1kg daily gain total requirements are 10p100 higher in 2 years old than in yearling of 450kg and 400kg BW respectively. Maintenance and growth requirements are both concerned: +9 and +12p100 respectively. At 425 kg BW total requirements increase by +10p100 as far as daily gain rise from 0.2 to 0.5kg for growth requirements rise by +300p100.

In the Netherlands according to CVB 1996 (Centraal Veevoeder Bureau), current Dutch growth rate recommendations do not distinguish between final adult weights and growth rates for various size horses and, therefore, might be overestimating the energy requirements of larger horses. In the CVB (1996) average daily gain is calculated according to p100 of BW, as taken from the growth curve devised in the late 1980's at PR (Bruin and Creemers, 1994) (Table 5). The formula to calculate fat content of the animal at each stage is referenced as GEH, 1982 – but is in fact derived from NRC (1978). From this premise the average daily gain in kg via the fat-gain and the protein-gain are calculated and finally the energy (in VEP = also a net energy unit) and protein (in VREp = digestible crude protein unit) requirements are determined:

1. Fat gain (FG; %) =  $0.1388 p + 1.111$  (from NRC, 1978)  
Protein gain (PG; %) =  $0.22 \times (100 - FG)$

where: p = p100 of adult weight at point in age (Table 5)

2.  $VEP_{\text{Growth}} = (0.03931 \times FG/0.75 + 0.02294 \times PG/0.45)/9.1414 \times 1000$

where: 0.75 = efficiency factor for fat gain  
0.45 = efficiency factor for protein gain (CVB, 1996)

3.  $\text{Total NE (VEP/day)}_{\text{Growth}} = \text{Maintenance}_{\text{Growth}} (\text{VEP}) + \text{Growth (VEP)}$

where:  $\text{Maintenance}_{\text{Growth}} (\text{VEP})$  (to 6 mths) (VEP) =  $47 \times BW^{0.75}$   
 $\text{Maintenance}_{\text{Growth}} (\text{VEP})$  (to 12 mths) (VEP) =  $44 \times BW^{0.75}$   
 $\text{Maintenance}_{\text{Growth}} (\text{VEP})$  (from 18 mths) (VEP) =  $42 \times BW^{0.75}$

*Table 5. Bodyweights of horses at various growth stages, p100 of adult weights, fat content and protein content per kilo weight gain (CVB, 1996).*

| Age in months             | 0              | 3   | 6   | 12  | 18  | 24  |
|---------------------------|----------------|-----|-----|-----|-----|-----|
| p100 of adult bodyweights | 10             | 30  | 47  | 68  | 82  | 89  |
| Adult weight              | Bodyweight, kg |     |     |     |     |     |
| 100                       | 10             | 30  | 47  | 68  | 82  | 89  |
| 200                       | 20             | 60  | 94  | 135 | 165 | 178 |
| 300                       | 30             | 90  | 140 | 202 | 247 | 267 |
| 400                       | 40             | 120 | 187 | 270 | 329 | 356 |
| 500                       | 50             | 150 | 234 | 337 | 411 | 445 |
| 600                       | 60             | 180 | 281 | 405 | 494 | 534 |
| 700                       | 70             | 210 | 327 | 472 | 576 | 623 |
| 800                       | 80             | 240 | 374 | 540 | 658 | 712 |
| Fat/kg ADG                |                | 67  | 133 | 195 | 234 | 257 |
| Protein/kg ADG            |                | 205 | 191 | 177 | 169 | 164 |

The graph (Figure 5) below shows the predicted growth curve of foals with adult weight of 500kg and 600 kg as currently estimated by the CVB (1996) and for comparison the growth curve as currently estimated by the GEH (1994). The gap between systems widens as adult bodyweight increases.

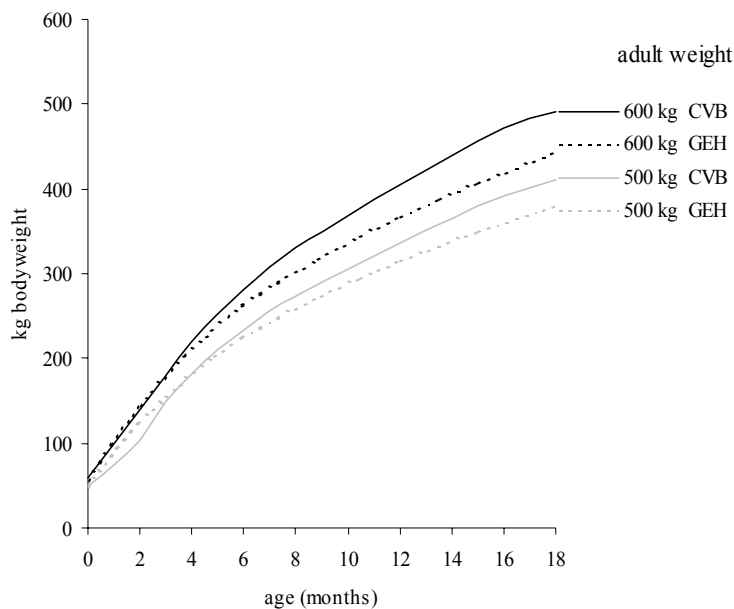


Figure 5. Growth curve of a 500 kg (grey) and 600kg (black) Warmblood foal up to 18 months as predicted by CVB (1996) and GEH (1994).

From point of weaning at 6 months to the one year old the NE (VEP) requirements per kilogram of gain (day) increase by 11p100. This means that at two years the young horse requires not much more than before at 11 p100 more energy to gain an extra kilo of BW compared to the 6 months old (Table 6). In the Dutch system these differences are relatively small as the percentage of final bodyweight plays the greatest role in determining requirements and the relationship between fat content and protein content within gained weight is taken as static throughout the growth period.

Table 6. variation with age of energy requirements per kg daily gain (CVB, 1996).

| Age                    | BW (kg) | NE (VEP)<br>requirements<br>for 1 kg<br>ADG | Total NE (Mcal)<br>requirements<br>for 1 kg<br>ADG | Increase p100<br>from weanling<br>value |
|------------------------|---------|---------------------------------------------|----------------------------------------------------|-----------------------------------------|
| Weanling, 6 months     | 234     | 1717                                        | 3.8                                                | 100                                     |
| Yearling               | 350     | 1900                                        | 4.2                                                | 111                                     |
| Long yearling, 18 mths | 411     | 1913                                        | 4.2                                                | 111                                     |
| 2 years old            | 445     | 1923                                        | 4.2                                                | 111                                     |

For 1kg daily gain, total requirements are 26p100 higher in yearling of 350kg BW than in weanling of 250kg BW). The increase deals with the maintenance and growth requirements: +20p100 and + 8p100 respectively. Comparing 1kg versus 0.5 kg daily gain in the yearling of 300kg BW, total requirements do not increase.

For 1kg daily gain, total requirements are 7p100 higher in two years old than in yearling of 450 and 400kg BW respectively. Maintenance and growth requirements are both concerned: +9p100 and +4p100 respectively. At 425kg BW, total requirements do not increase with variation of ADG: 0.5 vs 0.2kg.

In Scandinavian countries (1996-2002) requirements are calculated by using the standard growth curve for all types of horses defined by CVB (1996) (see Table 5), a similar model performed by INRA for estimating maintenance requirements and a model derived from NRC (1989) model for determining growth requirements. The various countries use either NE or ME systems and base energy requirements on average daily gain and age in months:

Country

DK, N, IS      UFC (NE) =  $ADG (1350 + 67.94 \text{ Age} - 1.093 \text{ Age}^2)/1000$

Sweden      MJ (ME) =  $ADG (1350 + 67.94 \text{ Age} - 1.093 \text{ Age}^2)13.45/1000$

Finland      F.U. (ME) =  $ADG (1350 + 67.94 \text{ Age} - 1.093 \text{ Age}^2)13.45/11700$

where: ADG = average daily gain

Age in months

Maintenance energy requirements are higher in very young foals than in 3 years old: + 12 p.100 but only 5 p.100 higher in yearling than 3 years old. For 1kg daily gain requirements for growth increase by 17 to 37 p100 in the yearling and 2 years old as far as the weanling is to be the reference (Table 7).

*Table 7. Maintenance requirements- Variation of energy requirements with age per kg daily gain (SCAND 2002).*

| Age     | Maintenance (kgBW <sup>0.75</sup> ) |         |           | Growth (for 1kg ADG) |         |           |      |
|---------|-------------------------------------|---------|-----------|----------------------|---------|-----------|------|
|         | UFC                                 | MJ (ME) | F.U. (ME) | UFC                  | MJ (ME) | F.U. (ME) | p100 |
| 0 - 6   | 0.047                               | 0.63    | 0.054     | 1.7                  | 23.11   | 1.97      | 100  |
| 7 - 12  | 0.044                               | 0.59    | 0.050     | 2.0                  | 27.00   | 2.31      | 117  |
| 12 - 18 | 0.042                               | 0.57    | 0.048     | 2.2                  | 29.84   | 2.55      | 129  |
| 18 - 24 | 0.042                               | 0.57    | 0.048     | 2.3                  | 31.62   | 2.70      | 137  |

For 1kg of daily gain total requirements are 19 p100 higher in weanling of 250 kg BW than in yearling of 350 kg BW. The increase deals with maintenance and growth requirements: + 20 and +17 p100 respectively. Comparing 1kg versus 0.5 kg daily gain in yearling of 300 kg BW total requirements rise by 23 p100 whereas growth requirements increase by 191 p100. For 1kg daily gain total requirements are 8p100 higher in 2 years old than in yearling of 450kg and 400kg BW respectively. Maintenance and growth requirements are both concerned: +9 and + 6p100 respectively. At 425 kg BW total requirements increase by +16p100 as far as daily gain rise from 0.2 to 0.5kg for growth requirements increase by +256p100.

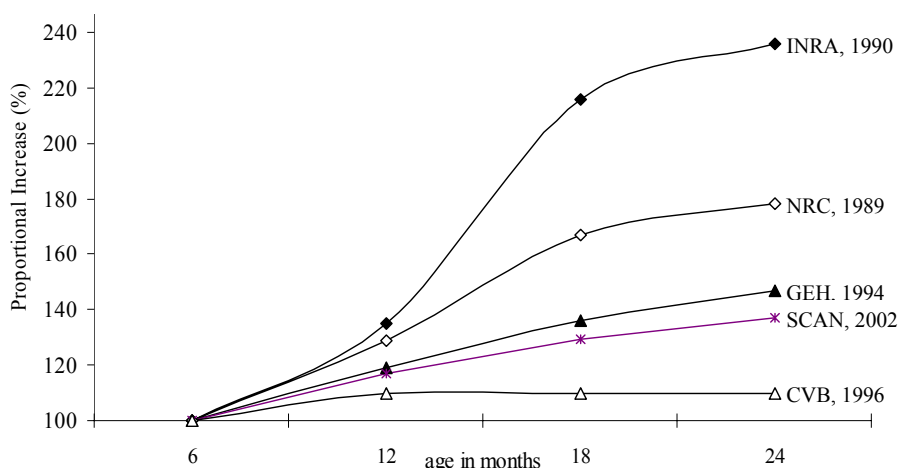
## Comparison of variations of energy requirements for growth with age, daily gain and body weight

In Table 8 the proportional increase with age in energy requirements per 1 kg ADG, with the requirements at 6 months set as a reference point, is shown for the different systems. Figure 6 highlights the differences in curvilinear relationships.

*Table 8. Comparison of variations of energy requirement for growth with age per 1 kg ADG across the different systems.*

|                        | NRC<br>(1989) | INRA<br>(1990) | GEH<br>(1994) | CVB<br>(1996) | SCAN<br>(2002) |
|------------------------|---------------|----------------|---------------|---------------|----------------|
| Weanling, 6 months     | 100           | 100            | 100           | 100           | 100            |
| Yearling               | 141           | 135            | 119           | 111           | 117            |
| Long yearling, 18 mths | 167           | 216            | 136           | 111           | 129            |
| 2 years old            | 178           | 237            | 147           | 111           | 137            |

In the Dutch system (CVB, 1996) energy requirements per kilo daily gain do not increase after 12 months of age. Requirements of energy per kg of daily gain rise continuously in NRC, GEH, and Scandinavian estimations accordingly a model whose slope is steeper for NRC estimations than in GEH and Scandinavian estimations. Variation of energy per kg daily gain is rising in INRA system according to a model where energy requirement per kg gain increase sharply at 18 months due to the coefficients b of the two nutritional models (Table 3 and Figure 6). This is consistent with the variation in chemical composition of daily empty body weight (EBW) (Figure 1). Lipids content in EBW rises sharply: +5% e.g.; +28p100 between 300 and 400kg EBW (e.g. 18- 24months ) whereas protein content is still very high: 20-19%. As a result energy requirements for the deposition of 1g of fat is known to be 7 times more expensive than the deposition of 1g of fat free-mass in growing cattle( Robelin and Daenicke, 1979). This is also due to the lower efficiency of conversion of metabolisable energy for protein accretion than for lipogenesis, a fact well known in growing cattle too (Kielanowski, 1965; Thorbek, 1972).



*Figure 6. Variation of energy requirements with age per kilo daily gain expressed as % of requirements (kg ADG) at 6 months of age (=100).*

Energy requirements for 1kg of gain from 6 months to 18 months old rise much higher in NRC and INRA estimations + 78 and 137 p100 respectively than in GEH, CVB and Scandinavian estimations: +11,+37 to 47 p100 respectively (Table 8). The steep rise within the INRA (1990) curve is due to the above explained change in distribution of fat and protein gain within the one kilo. This and the link to metabolic BW accounts for a sigmoidal curve development. For the NRC and the Scandinavian systems the increase in proportional energy requirements per kg gain per day is related to gain per day and age only, thus the current bodyweight does not influence calculations. In the GEH and CVB systems P100 of final bodyweight has been calculated into the equation, and the larger the percentage of final bodyweight reached, the lower the necessary increase in energy requirements, which explains the early flattening of their curves. This means once again that a direct comparison between energy requirements as attempted above is not strictly correct, but nevertheless highlights the different approaches within the systems. Comparing NRC and INRA estimations, energy requirements rise similarly until yearling stage: +41 and 35 p100 respectively; then requirements increase much higher in long yearling and 2 years old in INRA estimations: +116 and 137 p100 than in NRC estimations: +67 and 78 p100 (Table 8).

For 1 kg of daily gain at 250 and 350 kg BW, total energy requirements for all the systems rise between + 19 and 32 p100. But NRC and INRA variations are very close: 32 and 31 p100 respectively. The increase in total requirements is as much due to a rise in maintenance as due to a rise in growth requirements. For 1 kg of daily gain at 400 and 450 kg BW total energy requirements elevation range between +8 to 23 p100. Increase is much higher in NRC estimations: + 23p100 than in all the other estimations. Increase in maintenance and growth requirements are both concerned in all the estimations.

At 300kg BW increase in total energy requirements with daily gain: 0.5 to 1.0 kg, is very high in NRC estimations: +41 p100 and limited in other estimations (GEH, INRA, CVB, Scandinavia): 11 to 23 p100. The increase is of course due to the rise in growth requirements which range from 186 to 200 p100 in all estimations except in CVB calculations.

At 425 Kg BW increase in total requirements with daily gain: 0.2 to 0.5kg, is very high is NRC estimation +32p100 and limited in other estimations by GEH, INRA, CVB and Scandinavia +9 to +16p100, except for CVB. The increase is due to the rise in growth requirements which range from 246 to 300 p100 in all the estimations except for CVB.

### **Recommended allowances for energy in different countries**

In USA, NRC 1989 recommended allowances are drawn directly from requirements using the model set up. They are provided in tables designed for equines reaching different adult weights: 200 to 900 kg and between 4 and 24 months (Table 9).

Two levels of growth and corresponding recommended allowances are proposed to avoid bone disorders associated to high intake in carbohydrates (Glade and Belling, 1986; Savage *et al.*, 1993). In addition recommended allowances proposed in long yearling and 2 year olds are different as far as the horses are in or not in training. Energy requirement for training correspond to moderate work (+ 50 % maintenance DE).

*Table 9. Daily nutrients requirements and allowances of growing horses (500 kg mature weight) NRC 1989.*

|                          | Weight<br>(kg) | Daily gain<br>(kg) | DE<br>(Mcal) | CP<br>(g) | gCP/<br>Mcal DE | Lysine<br>(g) |
|--------------------------|----------------|--------------------|--------------|-----------|-----------------|---------------|
| Weanling 4 months        | 175            | 0.850              | 14.4         | 720       | 50              | 30            |
| Weanling 6 months        |                |                    |              |           |                 |               |
| Moderate growth          | 215            | 0.650              | 15.0         | 750       | 50              | 32            |
| Rapid growth             | 215            | 0.850              | 17.2         | 860       | 50              | 36            |
| Yearling 12 months       |                |                    |              |           |                 |               |
| Moderate growth          | 325            | 0.500              | 18.9         | 851       | 45              | 36            |
| Rapid growth             | 325            | 0.650              | 21.3         | 956       | 45              | 40            |
| Long yearling            |                |                    |              |           |                 |               |
| Not in training          | 400            | 0.350              | 19.8         | 893       | 45              | 38            |
| In training              | 400            | 0.350              | 26.5         | 1195      | 45              | 50            |
| Two years old, 24 months |                |                    |              |           |                 |               |
| Not in training          | 450            | 0.200              | 18.8         | 800       | 43              | 32            |
| In training              | 450            | 0.200              | 26.3         | 1117      | 43              | 45            |

DE = Digestible Energy

CP = Crude Protein

In Germany, GEH 1994 recommended allowances are drawn directly from requirements. They are provided in tables designed for equines reaching 100 to 800 kg adult body weight and between 6 and 36 months of age (Table 10). Not any particular growth level is specified but 20% additional energy for physical activities are included in maintenance requirements for all ages.

*Table 10. Daily nutrient requirements of growing horses (500 kg mature weight) – GEH, 1994.*

|                                | Weight<br>(kg) | DE<br>Mcal | DCP<br>(g) | g DCP/<br>Mcal DE | Lysine<br>g/Mcal DE |
|--------------------------------|----------------|------------|------------|-------------------|---------------------|
| Weanling (3 – 6 months)        | 125 – 225      | 15.1       | 580        | 38                | 2.1                 |
| Yearling (7 – 12 months)       | 225 – 315      | 15.8       | 540        | 34                | 1.9                 |
| Long Yearling (13 – 18 months) | 315 – 380      | 16.3       | 485        | 30                | 1.9                 |
| 2 years old* (19 – 24 months)  | 380 – 425      | 16.7       | 445        | 27                | 1.9                 |
| 3 years old* (25 – 36 months)  | 425 – 475      | 17.7       | 415        | 23                |                     |

DE = Digestible Energy

DCP = Digestible Crude Protein

\* not in training

In France, INRA (1990) recommended allowances have been calculated using the 2 models set up for light breeds or heavy breeds and the growth curves drawn from feeding experiments (Figure 7). The recommended allowances are provided in tables designed for horses reaching 400 to 900 kg body weight and between 6 and 36 months of age (Table 11).

Table 11. Daily nutrients requirements (1) and allowances of growing horses (500 kg mature weight) – INRA, 1990.

|                             | Weight<br>(kg) <sup>5</sup> | Daily gain<br>(kg/d) | NE  |       | MADC<br>(g) <sup>3,4</sup> | g MADC |       |
|-----------------------------|-----------------------------|----------------------|-----|-------|----------------------------|--------|-------|
|                             |                             |                      | UFC | Mcal  |                            | /UFC   | /Mcal |
| Weanling (6 months)         | 220                         | 0.800                | 4.2 | 9.24  | 560                        | 133    | 61    |
| Yearling (6–12 months)      |                             |                      |     |       |                            |        |       |
| Optimal growth              | 320                         | 0.700-0.800          | 5.5 | 12.10 | 590                        | 107    | 49    |
| Moderate growth             | 280                         | 0.400-0.500          | 4.5 | 9.90  | 440                        | 98     | 44    |
| 2 years old* (18–24 months) |                             |                      |     |       |                            |        |       |
| Optimal growth              | 470                         | 0.400-0.500          | 6.8 | 14.96 | 420                        | 62     | 28    |
| Moderate growth             | 440                         | 0.150-0.200          | 6.0 | 13.20 | 320                        | 55     | 24    |
| 3 years old* (30-36 months) |                             |                      |     |       |                            |        |       |
| Optimal growth              | 490                         | 0.150-0.250          | 6.5 | 14.30 | 330                        | 51     | 23    |
| Moderate growth             | 470                         | 0 – 0.100            | 6.0 | 13.20 | 260                        | 43     | 20    |

NE = Net Energy; UFC = Horse Feed Unit

MADC = Horse Digestible Crude Protein

\* not in training, but to be added if training:

- + 1.0 – 1.5 UFC/d for light work
- + 2.0 – 2.5 UFC/d for moderate work

<sup>1</sup> no feeding experiments were conducted where the young horses are: 12-18 months, 24-30 months as far as the young horses are grazing in French conditions, but requirement could be estimated by interpolation.

<sup>2</sup> medial BW during the period

<sup>3</sup> lysine (%TDMI): 0.6; 0.5; 0.4% for weanling, yearling and 2-3 years old respectively (according to NRC, 1989)

<sup>4</sup> threonine(%TDMI): 0.5; 0.4% for weanling and yearling respectively (according to Ott, 2001)

They have been extensively validated in long term feeding experiments conducted either with light breeds (sports horses: Anglo Arabian – Selle Français) or with heavy breeds. (see synthesis of Micol and Martin-Rosset, 1995). Two levels of allowances are suggested for light breeds according to the goal of production: horse produced for competition vs leisure and the growth potential of the young horses. The recommended allowances are calculated either for an optimum growth or for a moderate growth accordingly to growth curves which have been established by implementing long term feeding experiments (Figure 7).

Optimum and moderate growth are suggested as it as been observed at INRA that bone disorders were associated with maximum growth and body development closely linked with body weight increase and with a strong interaction with body weight at weaning (Trillaud-Geyl *et al.*, 1992; Martin-Rosset, 2001). Young horses managed accordingly to a moderate growth between 6 to 40 months are capable to reach similar body development no much later than those conducted with optimal growth as far as young horses are able to have compensatory growth during summer season (Bigot *et al.*, 1987; Trillaud-Geyl *et al.*, 1992).

For young horse in training, additional energy allowances due to exercise have to be supplied accordingly to appropriate work intensity (INRA, 1990). Extra energy allowances are designed jointly with extra protein allowances due to muscle development (see protein).



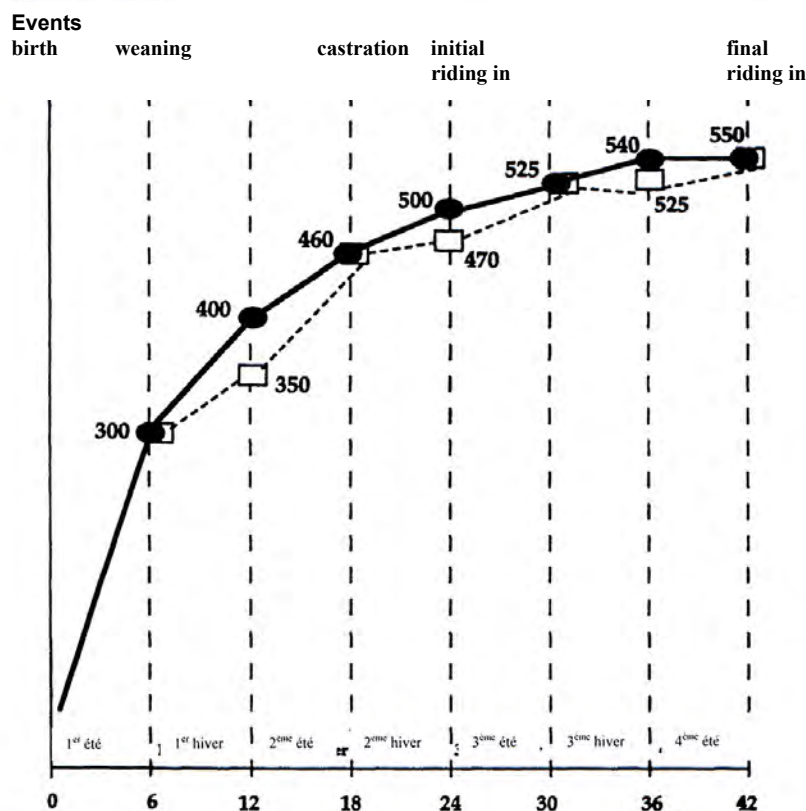


Figure 7. Growth curve management of young sport horses (selle Français or anglo-Arab) established from INRA long term feeding experiments (where ● early horses; and □ late horses; INRA, 1990).

In the Netherlands, CVB (1996) energy allowances are drawn directly from requirements (Table 12). Daily gain is estimated at 'optimum' level according to the above described growth curve (proportion of adult weight reached at certain age (Figure 5). For a horse to reach 500 kg in bodyweight, this leads to the following recommendations:

Table 12. Recommended growth rates and energy allowances for a 500 kg sports horse(CVB, 1996).

| Age | % of final BW | growth/day (kg) | VEP  | VREP |
|-----|---------------|-----------------|------|------|
| 3   | 30            | 1.093           | 3640 | 650  |
| 6   | 47            | 0.751           | 4150 | 530  |
| 12  | 67            | 0.478           | 4420 | 425  |
| 18  | 82            | 0.287           | 4460 | 380  |
| 24  | 89            | 0.164           | 4450 | 350  |
| 30  | 94            | 0.109           | 4500 | 340  |
| 36  | 97            | 0.082           | 4540 | 340  |

VEP = Dutch Feed Unit (compare to UFC by VEP/1000)

VREP = Dutch Protein Unit = Digestible Crude Protein (directly comparable to DCP-GEH)

However, a recent review studying the growth curves of 144 Dutch Warmblood foals in relation to their bone development (osteochondrosis) has recommended to reduce energy allowances for animals from 500 kg onwards to daily growth similar to the OC negative group (Ellis and Van Tilburg, 2002). This would mean that the p100 adult bodyweight at set age points decreases accordingly. Table 13 below summarises the adjusted percentages of final body weight and Figure 8 shows the suggested new growth curve.

Table 13. Suggested weights of growing horses as p100 of final weight.

|              | Age in months        |    |    |    |    |
|--------------|----------------------|----|----|----|----|
|              | 0                    | 3  | 6  | 12 | 18 |
| Adult weight | p100 of final weight |    |    |    |    |
| 100-400Kg    | 10                   | 30 | 47 | 68 | 82 |
| 500          | 10                   | 29 | 44 | 64 | 78 |
| 600          | 10                   | 29 | 43 | 62 | 75 |
| 700-800      | 10                   | 27 | 41 | 59 | 74 |

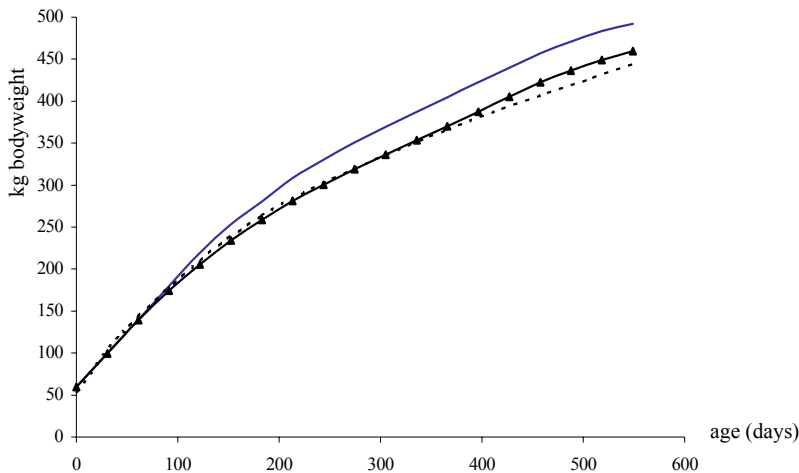


Figure 8. Growth curve of a 600kg Warmblood foal up to 18 months of age as predicted by CVB (1996, black line) and GEH (1994, punctuated line) and with new proposed Growth Curve for the Dutch System (Ellis and Van Tilburg, 2002, line and triangles).

In terms of p100 adult weight the change seems small but leads to a noticeable reduction in energy allowances from 3 months to 6 (e.g. 600 kg horse – reduction of 300 VEP/d e.g. 6p100) and up to 12 months. Alternatively it was recommended that the daily gain and weight changes could be recorded as two separate columns, with one referring to slow growth and one to fast growth, as in NRC (1989) and INRA (1990). An adjustment for horses under 400 kg has not been made, as these will reach their final bodyweight at an earlier stage than larger animals. Further research into whether it is necessary to adjust the bodyweight of smaller horses at set stages is necessary.

In Scandinavia, recommended allowances are drawn directly from the calculation of requirements using the different models set up and the growth curve drawn from CVB (1996) (Table 14).

*Table 14. Daily nutrients requirements of growing horses (500 kg mature weight) Scandinavian countries (adapted from Austbø, 2004).*

|                         | Weight<br>(kg) | Daily gain<br>(kg) | Energy           |                      | Protein         |     | N/E |     |     |
|-------------------------|----------------|--------------------|------------------|----------------------|-----------------|-----|-----|-----|-----|
|                         |                |                    | UFC <sup>1</sup> | ME <sup>2</sup> (MJ) | Fu <sup>3</sup> | DCP | UFC | ME  | Fu  |
| Weanling (6 months)     | 235            | 0.765              | 4.1              | 55.5                 | 4.7             | 530 | 129 | 9.5 | 113 |
| Yearling (12 months)    | 335            | 0.464              | 4.4              | 58.7                 | 5.0             | 425 | 97  | 7.2 | 85  |
| 2 years old (24 months) | 460            | 0.164              | 4.6              | 9.8                  | 5.20            | 350 | 76  | 5.7 | 67  |
| 3 years old (36 months) | 485            | 0.055              | 4.5              | 9.0                  | 5.1             | 350 | 76  | 5.7 | 69  |

UFC = Horse Feed Unit  
 DCP = Digestible Crude Protein  
 ME = Metabolisable Energy  
 Fu = Feed unit

<sup>1</sup>Denmark, Island, Norway  
<sup>2</sup>Sweden  
<sup>3</sup>Finland

### Comparison of requirements in relation to allowances for energy

As far as total requirements are concerned and compared at each age (12, 24, 36 months) in each country relatively to requirements at 6 months old the following can be found (Table 15).

*Table 15. Comparison of the relative variation in total energy (E) and nitrogen (N) requirements for a growing horse to make 500 kg adult weight for ADG suggested at each age in each country.*

| Country /Institution |     | Age |     |         |     |
|----------------------|-----|-----|-----|---------|-----|
|                      |     | 6   | 12  | 24      | 36  |
| USA/NRC (1)          | E   | 100 | 125 | 117 (2) | -   |
|                      | N   | 100 | 112 | 99 (2)  | -   |
|                      | N/E | 100 | 90  | 90(2)   | -   |
| Germany/GEH          | E   | 100 | 105 | 111     | 117 |
|                      | N   | 100 | 93  | 84      | 72  |
|                      | N/E | 100 | 89  | 79      | 71  |
| France/INRA (1)      | E   | 100 | 110 | 126     | 120 |
|                      | N   | 100 | 92  | 66      | 53  |
|                      | N/E | 100 | 77  | 44      | 35  |
| The Netherlands/CVB  | E   | 100 | 107 | 107     | 109 |
|                      | N   | 100 | 80  | 66      | 64  |
|                      | N/E | 100 | 75  | 61      | 58  |
| Scandinavia          | E   | 100 | 106 | 108     | 108 |
|                      | N   | 100 | 80  | 66      | 66  |
|                      | N/E | 100 | 75  | 59      | 59  |

<sup>1</sup>Average growth =  $\frac{\text{rapid} + \text{moderate}}{2}$

<sup>2</sup>not in training

Energy requirements increase between 5 to 26 % in yearling, 2 or 3 years old in all the countries. The increase is higher namely for the yearling in NRC estimation( + 25 p100) than in other countries (+5 to 10 p100). The increase is higher for the 2 years old in France (+ 26 p100) than in the other countries ( + 11 to 18 p100). At 3 years of age the increase is very close in all the European countries.

As discussed in the introduction actual feed recommendations and allowances per country do also involve the amount of energy supplied by a feed and as such this calculation cannot be completely separated from requirements. In order to incorporate these we looked at application tables and without converting the various units used in each country we simply calculated the requirements for a foal with adult weight of 500 kg at medium growth and expressed these in DM Barley units as they are given by each system (Figure 9). Expressed as barley units per country (kg DM barley) the results are very similar to the proportional changes calculated in Table 15.

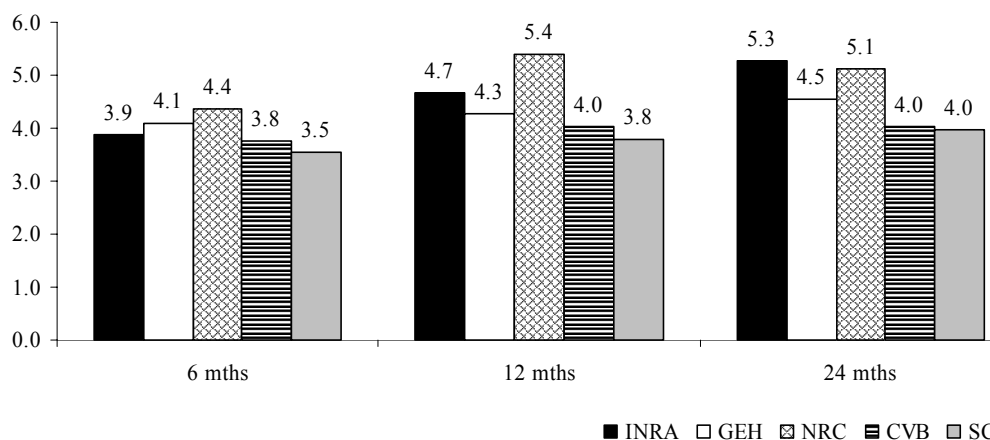


Figure 9. Comparison of energy intake expressed in kg DM barley calculated per system for a foal to make 500 kg adult weight at medium growth.

## Evaluation of protein requirements and allowances

### Current protein evaluation for growth in different countries: data base – methodology - requirements

In USA according to NRC 1989 protein requirements for maintenance would be 1.3 g CP/kg BW/day assuming that a value of 0.60 g DCP/kg BW/day appears to be appropriate for most horses and if the horse consumes a forage diet with a digestibility of 46 p.100. Dividing the crude protein requirements by the Digestible Energy requirements results in a value of 40 g CP/Mcal DE. Adequate essential amino acids (EAA) requirements are met when horses are fed typical feedstuffs to meet proteins requirements. Based on available experimental data obtained in growing horses (Wirth *et al.*, 1977; Meakin, 1979; Ott *et al.*, 1979; Potter, 1981; Ott and Asquith, 1983, 1986; Boren *et al.*, 1987; Gibbs *et al.*, 1987; Scott *et al.*, 1987; Secureketal, 1987), CP requirement for growth were calculated to be 50, 45 and 42.5g CP/Mcal DE/d for weanlings, yearlings and 2 years old respectively.

Lysine requirements are 2.1; 1.9 and 1.7 g/Mcal DE/day at the same reference ages (Breuer *et al.*, 1970; Breuer and Golden, 1971; Hintz *et al.*, 1971; Potter and Huchton, 1975; Ott *et al.*, 1979, 1981; Ott and Asquith, 1986).

For 1kg of daily gain, total protein requirements are 31 p100 higher in yearling of 350 kg BW than in weanling of 250 kg BW. Maintenance and growth requirements increase by + 40 and +26 P100 respectively. Comparing 1kg to 0.5kg daily gain in the yearling of 300kg BW total protein requirements increase by +53p100 whereas growth requirements rise by +200 p100. For 0.5kg daily gain total protein requirements are +10p100 higher in the 2 years old of 450kgBW than in the long yearling of 400kgBW. Maintenance and growth requirements are both concerned: +13 and + 7 p100 respectively. At 425kg BW total requirements increase by +34p100 as far as daily gain rises from 0.2 to 0.5kg, for growth requirements increase by +250p100.

In Germany, according to GEH 1994 protein requirements for maintenance were calculated at each age and body weight using 3 g DCP/kg BW<sup>.75</sup> to be added to growth requirements. Protein requirements for growth were calculated using a growth model (see energy) and the composition of the weight gain in the young horse drawn from a calculation using equation of NRC 1978 for fat and protein and based on the utilisation of digestible protein for growth is 65 p.100 up to 6 months of age then 35p100 for older young horses:

body fat % = 0.1388 x relative body weight (proportion of adult weight) + 1.11

body protein % = 0.22 x (100 – body fat)

Protein/energy requirements ratio range from 38 g DCP/Mcal DE in weanling to 23 DCP/Mcal DE in 3 years old. Lysine requirements are those proposed by NRC 1989.

For 1kg daily gain, total requirements are 20p100 higher in yearling of 350kg BW than in weanling of 250kg BW. Maintenance and growth requirements increase by +4 and + 33 p100 respectively. Comparing 1kg to 0.5 kg daily gain in the yearling of 300kg BW total requirements increase by + 46p100 whereas growth requirements rise by + 200p100. For 0.5kg daily gain total requirements are +4p100 higher in the 2 years old of 450 kg BW than in the long yearling of 400kg BW. For maintenance only requirements are +9p100 higher. At 425kg BW total requirements increase by +33p100 as far as daily gain rise from 0.2 to 0.5kg BW, for growth requirements increase by +250 p100.

In France according to INRA 1990 heavy breeds and light breeds were distinguished due to experimental data available in addition to their own characteristics linked to specificity of the heavy breeds.

Protein requirements for maintenance were estimated to be 2.4 g DCP (in French MAD)/kg BW<sup>.75</sup> referring to nitrogen balance carried out by Slade *et al.* (1970) and Prior *et al.*, (1974) and is, expressed in MADC (translated as: corrected DCP). In the MADC system, protein value of the feeds account for the true amount of AA absorbed in the small and large intestine. As far as concentrates are mainly digested in the small intestine there is no difference between DCP and MADC. Contrary the difference between DCP and MADC might reach on average 10 to 35p100 because forages are digested both in small and large intestine. But the latest fraction does not provide significant proportion of AA absorbed. As a result the requirements expressed in DCP have to be increased by 10 to 20p100 to be expressed in MADC as young horses are fed mixed diets (Martin-Rosset *et al.*, 1994). This means requirements are 10-15

p100 higher: 2.8 g MADC/kg BW<sup>.75</sup>, since DCP content of forages had been reduced by 10-20 p.100 to be expressed in MADC (Martin-Rosset *et al.*, 1994). Maintenance MADC requirements are 70 g MADC/UFC or 32 g MADC/Mcal NE.

Protein requirements of growing horses (above maintenance) were calculated from protein retention. The latter can be determined accurately from the composition of empty body weight and weight gain. This was defined on the bases of anatomical composition data obtained of heavy breeds slaughtered at different ages (Martin-Rosset *et al.*, 1983b) after several feeding trials. Protein requirements of heavy breeds were estimated by the factorial method because all the feeding trials were designed to define the optimum CP content of the diet or the effect of feed protein quality on the growth of the young horses (Agabriel *et al.*, 1984). The efficiency of MADC stated was 45 p.100 as suggested by NRC (1978). 25 p.100 were added to the estimated requirements until one year of age to take into account the faster turn-over of body protein as measured in other species (Reeds and Harris, 1982).

In light breeds protein requirements were calculated by using a mathematical model similar to that used for energy as there were enough INRA experimental trials designed to study the effect of CP content of the diet on average daily gain (Bigot *et al.*, 1987; Trillaud-Geyl *et al.*, 1992):

gMADC/day = a BW<sup>.75</sup> + b G  
 where: BW<sup>.75</sup> = Metabolic Body Weight  
           G = Daily Gain (kg)  
           a = maintenance requirements  
           b = coefficient linked to age (Table 16)

Table 16. Values of the coefficient a and b for calculating MADC requirements for light breeds (INRA, 1990).

| Ages (months) | a   | b   |
|---------------|-----|-----|
| 6-12          | 3.5 | 450 |
| 18-24         | 2.8 | 270 |
| 30-36         | 2.8 | 270 |

Maintenance requirements arise to be 3.5 g/kg BW<sup>0.75</sup> in yearling, then 2.8 g/kg BW<sup>0.75</sup> in 2 and 3 years old; as a result there are similar to maintenance in the adult for the latest (Table 11). Gain is not affected by an exponent as for energy model as allometry of muscles and derived protein composition do not vary as much as adipose tissue and lipids content after weaning in breeding horses (not in training). However the variation is taken into account indirectly via b coefficient which affects Gain = 450 versus 270 in yearling and 2-3 years old (Table 16).As a result at a constant daily gain the intake of MADC rises with body weight until 350kg BW because, using the designed model, the intake of protein is proportional to BW<sup>0.75</sup> (0.75< 1) and then maintenance is more and more of major concern (Figure 10). At a constant body weight the ratio protein/kg daily gain rises as far as the daily gain increases but less and less because G is affected by a coefficient b which is linked to the limited amount of protein which is synthesised (Table 16). The protein/energy requirements ratio ranges from 103 g MADC/UFC or 47 g MADC/Mcal NE in yearling to 47 g MADC/UFC or 22 g MADC/Mcal NE in three years old horses.

For a constant daily gain, the intake of MADC increases more and more: CB << BA (0.3 kg ADG) then CB > BA (0.7 kg ADG) and CB >> BA (0.9 kg ADG) because in the designed model, protein intake is partially proportional to BW<sup>0.75</sup> (maintenance), whereas ADG is not affected by BW<sup>0.75</sup> as in the model for energy (Figure 10).

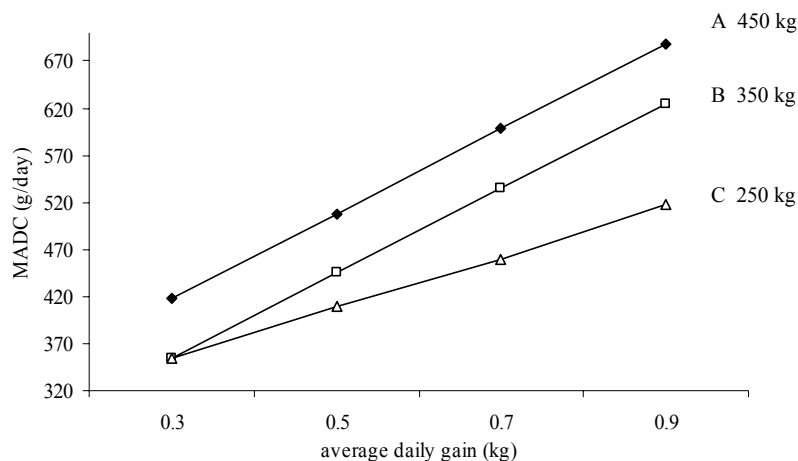


Figure 10. Variation in MADC requirements according to BW reached at 12 months and different average daily gains (Source: INRA, 1990).

Lysine requirements based on NRC 1989 requirements were calculated to be 0.6 0.5 0.4 p100 TDMI (Total Dry Matter Intake) in weanling, yearling and 2-3 years old respectively. Feed protein quality becomes less important as the growth potential decreases with age and the proportion of hay in the diet increases simultaneously.

For 1kg daily gain total requirements are 9p100 higher in the yearling of 350kg BW than in the weanling of 250kg BW; For maintenance only: requirements are +28p100 higher in the yearling of 350kg BW than in the weanling of 250kg BW. Comparing 1kg to 0.5kg daily gain in the yearling of 300kg BW total requirements are 54p100 higher whereas growth requirements rise. For 0.5kg daily gain total requirements are 6p100 higher in the 2 years old of 450 kg BW than in the long yearling of 400kg BW. Maintenance is only concerned: +10p100. At 425kg BW total requirements are 26p100 higher as far as the daily gain rise from 0.2 to 0.5kg, for growth requirements increase by + 250p100.

In the Netherlands, according to CVB (1996) protein requirements for maintenance from 7 months of age are calculated according to the GEH (1994) system, of 3 g DCP/kg BW<sup>0.75</sup>. Below 7 months of age an increased maintenance is calculated, according to INRA (1994), making the calculation 3.5g DCP kg BW<sup>0.75</sup>. To add protein requirements for growth the daily protein gain calculated as described above (Table 12), and utilised in GEH (1994), is used, assuming a conversion efficiency of DCP into body protein of 45 p100. This efficiency is deemed too low for young horses, but it was decided to keep to this factor within the current system (CVB, 1996):

$$\text{VREp}_{\text{Growth}} (\text{g/day}) = \text{PG}/0.45$$

where: PG = protein gain (g/day)

VREp = Digestible Crude Protein for horses

For lysine requirements it was decided to keep to factorial calculations from in vivo experimental results in pigs, where the net lysine requirements for maintenance were set at 36mg/kg BW<sup>0.75</sup> and the requirements for growth were set at 68 mg/g protein gain (Van der Peet Schwing *et al.*, 1994) For foals at 3, 6 and 12 months this comes to a total requirement of 6, 5 and 4.5g lysine respectively per 100g VREp requirements.

In Scandinavia, according to Austbø (2004) requirements for growth were calculated from a standard growth curve for all types of horses drawn from Dutch work (CVB 1996) and maintenance requirements were added (Table 14). Maintenance requirements are not different in growing horses to those in adult horses. Protein requirements for growth were calculated at different body weights using standard growth curve, protein disposition per kg weight gain (20 to 16% from birth to 3years of age) and DCP conversion rate of 45p100 (Table 14). Protein/energy requirements ratio are 128 g DCP/UFC, 9.5 g DCP/MJME or 111 g DCP/Fu in weanling expected to reach 500 kg adult body weight (Table 14).

Lysine requirements are those stated by CVB 1996: 5.8 – 5.0 and 4.5 g/100 g DCP for 3, 6 and 12 months old respectively.

For 1kg daily gain total requirements are 7p100 higher in the yearling of 350kg BW than in the weanling of 250kg BW. Maintenance is only concerned: +20p100. Comparing 1kg to 0.5kg daily gain total requirements are 68 p100 higher in the yearling of 300kg BW whereas growth requirements rise by +200p100. For 0.5kg daily gain total requirements are 6p100 higher in the 2years old than in the long yearling. Maintenance is only concerned: +9p100. At 425kg BW total requirements are 30p100 higher as far as the daily gain rise from 0.2 to 0.5kg for growth requirements rise by +250p100.

### Variation of protein requirements with age, daily gain and body weight

In Figure 11 the proportional increase with age in protein requirements per 1 kg ADG, with the requirements at 6 months set as a reference point, is shown for the different systems.

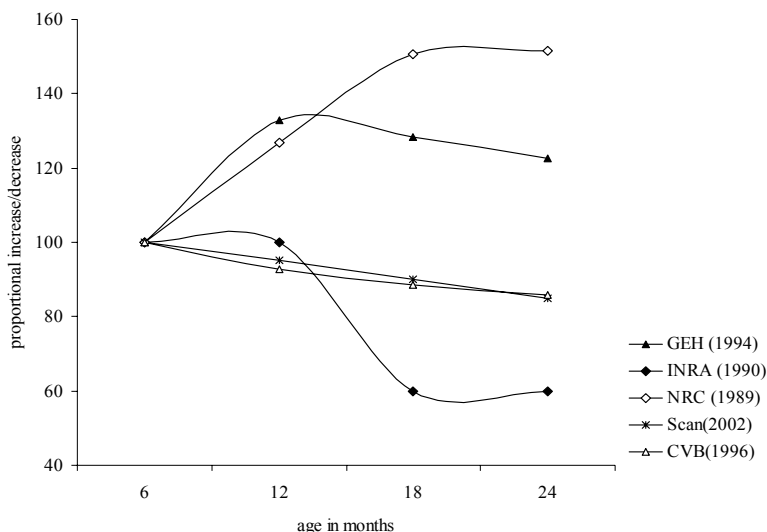


Figure 11. Variation of protein requirements with age per kilo daily gain expressed as p100 of requirements (kg ADG) at 6 months of age (=100).



Protein requirements per kg daily gain rise until 12 or 18 months of age in GEH and NRC estimations respectively then decrease. The GEH system decreases DCP requirements according to % of protein in gained body weight, while the NRC system links the CP intake directly to the decrease in energy required for average daily gain at that age. In the Scandinavian and the CVB system, requirements per kg of gain decrease continuously from 6 to 24 months of age. The difference between these two systems and the GEH system are caused only by difference in factors for the efficiency of conversion of feed protein to body protein. In the INRA system the protein requirement per kg of gain is steady from 6 to 12 months, then decrease significantly. This is consistent with the significant decrease of the protein content of daily empty body weight (EBW) at 350-400kg of empty body weight (12-24 months): -4% e.g. expressed in relative value to total protein content of EBW: -20p100 compared with the slight decrease between 6 and 12 months (200-300kg EBW): <1% e.g. 5p100 (see Figure 1).

For 1kg of daily gain at 250 and 350 kg BW the increase in total requirements is higher in NRC and GEH estimations (+31 and 20p100 respectively than in INRA, +9 to +7p100 than in CVB and Scandinavian estimation). Maintenance and growth requirements are both concerned in NRC estimations +40 and +26p100 respectively and in GEH estimations: +4 and 33p100 respectively. Maintenance requirements are only involved for INRA, CVB and Scandinavia: +20 to 28p100.

For 1kg of daily gain at 400 and 450kg BW total requirements are 4 to 10p100 higher in all the estimations but due to mainly to the increase in maintenance requirements: +9 to +13p100 except for NRC estimations where there is an increase in growth requirements too: +7p100. As far as the daily gain is very high the proportion of increase in maintenance requirements is as high as the body weight is low: 250 to 350kg vs 400 to 450kg. Total requirements rise between 46 to 68p100 at 300kg BW with increasing daily gain and between 26 to 33p100 at 425kg BW with increasing daily gain as far as the proportion of the increase in growth requirements in total requirements reach 200p100 and 250p100 at 300kg and 425kg BW respectively in all the estimations.

### **Recommended allowances for protein in different countries**

Recommended allowances for protein were listed in tables of recommended allowances for energy above (Tables 9 – 14).

In USA, NRC (1989) recommended allowances are the same as the requirements (Table 9). They are provided in tables designed for equines reaching different adult weight: 200 to 900 kg mature weight and between 4 to 24 months of age.

Two levels of growth and recommended allowances are proposed to avoid bone disorders (Table 9). In addition recommended allowances proposed in long yearling and 2 years old are different as far as the horses are in or not in training. Protein requirements for training correspond to moderate work (+ 50 % maintenance DE requirements + 40 g CP/extra Mcal DE over maintenance). Lysine recommended allowance are appropriate to the growth level (Table 9).

In Germany, GEH 1994 recommended allowances are the same as the requirements (Table 10). They are provided in tables designed for equines reaching different adult weight 100 to 800 kg mature weight and between 3 to 36 months of age.

In France, INRA 1990 recommended allowances are estimated from growth curve drawn from feeding experiments using the appropriate model (Table 16). Two levels of allowances are suggested as for energy considering the goal of production (Table 11).

In the Netherlands, CVB 1996 recommended allowances are the same as requirements (Tables 12 and 13) and these are given in tables from 6 – 36 months

In Scandinavian countries, according to Austbø (2004) recommended allowances are estimated using a growth curve of body weight calculated from the proportion of adult body weight, and derived daily gains at different ages and adult body weights.(100 to 800kg).

For practical purpose the protein/energy ratio for horses with 500 kg adult body weight can be used for calculating the allowances of all growing horses (Table 14).

### **Comparison of protein requirement or allowances**

As far as allowances are concerned and compared at each age (12, 24, 36 months) in each country relatively to requirements of weanlings it appears the main following figures are arising (Table 15).

In NRC recommendations, protein requirements increase by 12 p.100 in the yearling then keep on being steady in 2 years old not in training. Protein allowances do not increase significantly in the yearling and then decrease progressively by 16 to 28 p.100 in the 2 and 3 years old respectively in GEH estimations, by 34 and 47p100 in INRA recommendations respectively. In CVB protein requirements reduce by 20 p100 from weaning to 12 months, by 34 p100 and 36 p100 from weaning to 24 months and 36 months respectively. In Scandinavian recommendations proteins allowances decrease with age from 20 p100 in the yearling to 34p100 in the 2 and 3 years old.

### **Protein/energy ratio**

Protein/energy ratios (Table 15), expressed in each estimation relatively to weaning ratio, are lower in European than in American recommendations: 75-89 p100 vs 90 p100 in the yearling, 44-79 p100 vs 90 p100 in 2 years old respectively in Europe and in USA. At 3 years of age N/E ratio is very low in INRA and low in CVB and Scandinavian recommendations: 35 to 59 p100 compared to GEH recommendations: 71p100.

### **Amino acids requirements**

Lysine requirements are: 0.5% and 0.6% of total dry matter intake(TDMI) for weanling and yearling, two years old respectively (NRC, 1989). Such requirements are supported by recent work of Graham *et al.* (1994), Coleman *et al.* (1997) and Staniard *et al.* (1999). For threonine 0.5% and 0.4% TDMI are suggested for the weanling and the yearling respectively by Ott (2001). For methionine experimental data are contradictory (Borton *et al.*, 1973; Meakin *et al.*, 1979). Tryptophan is not likely to be limiting as tryptophan content of horse feedstuffs is high.

### **Effect of training on the requirements and allowances**

A greater supply of protein to the long yearling and the two years old should be necessary to enable increases in muscular mass, in the concentration of myoglobin and in enzymes

responsible for muscular metabolism and thus to prevent anaemia. Drawn from knowledge in human nutrition, protein supply should be increased by 20 p100 at the beginning of training and by 100 p100 during intensive training (Willmore and Freund Beau, 1984). There is no evidence to increase the protein or AA supplementation to equine over what is recommended for human athletes (see reviews of Lawrence, 1998; Martin-Rosset and Tisserand, 2004).

In USA, energy requirements of long yearling (18 months) and two years old in training are increased by 50% for exercise which is estimated to meet medium intensity in the NRC scale (NRC, 1989). As far as the weight of long yearling and the two years old does not change whether they are in training or not, and CP/DE ratios are the same for maintenance and work, total protein requirements increase by 34 p100 and 43p100 respectively.

In Germany, 20 p100 of additional protein (and energy) are included in the total requirements and allowances which are proposed by GEH (1994) to take in account the effect of additional natural movement of young horses during the growth period. For young horses in training no extra allowances are given and energy and protein allowances increase using the same calculation as is used for adult animals (for recommendations see Kienzle, 2004; Coenen, 2004). This leads to a mean increase in energy requirements for light training of 25 p100, medium training of 50 p100. Protein intake is recommended to be kept at the same ratio to protein intake at maintenance of 5 g DCP to 1MJ DE (e.g. 1.2gDCP/Mcal DE).

In France, 30, 60 or 90 p100 of additional energy and protein for long yearling, two and three years old horses in training should be implemented according to the intensity scale proposed by INRA: very light, light or medium exercise, as far as protein/energy ratio is 65g MADC/additional UFC (e.g. 31g MADC/additional Mcal NE) as for maintenance (INRA, 1990).

In The Netherlands for young horses in training no extra allowances are given and energy and protein allowances increase using the same calculation as is used for adult animals (for recommendations see Ellis, 2004). This leads to a mean increase in energy requirements for light training of 23 p100, medium training of 40 p100. Protein intake is recommended to be kept at the same ratio to protein intake at maintenance of 1VREP to 13 VEP.

In Scandinavian countries, we can presume that additional protein could be supplied to young horse in training according to the intensity of exercise designed in the NRC 1989 scale which is used Scandinavia, using the DCP / energy ratio of: 80g DCP/F.U. in Denmark, Norway and Island; 3gDCP/MJ ME in Sweden; and 70g DCP/ME (F.u.) in Finland.

## **Discussion and conclusion**

### **Variation of total energy or protein requirements with BW or ADG:**

Comparing the variations in percentage of total energy requirements with BW whatever the estimations at the same ADG appropriate to the age, total requirements rise with BW but more in the early age: yearling(350kg) vs Weanling (250kg): +19 to 32p100 than in the older age: 2years old (450 kg) vs long yearling(400kg): 8 to 23p100. Variation in maintenance and growth requirements contribute both to the total increase. Comparing the variation in percentage of total energy requirements with ADG at the same BW, total requirements rise but slightly more in the early age: +11 to +41p100 than in the older age: +9 to +32p100. Variation in growth requirements seems only to contribute to the total variation.

Comparing variation in percentage of total protein requirements at the same ADG, total requirements rise with BW but more in the early age: +7 to 31p100 than in the older age: +6 to 10p100. Variation in maintenance requirements is mainly involved. Comparing the variation in percentage of total protein requirements with ADG at the same BW, total requirements rise sharply but more in the early ages: +46 to 68p100 than in the older age: 26 to 36p100. Variation in growth requirements is of course involved. Variations in percentage of total requirements seem to be more affected by ADG than by BW namely for protein requirements and to a lesser extend for energy requirements.

**Effect of BW and ADG on maintenance/growth requirements ratio in total energy or protein requirements:**

The proportions of maintenance and growth requirements in total requirements have been compared between the different estimations proposed by the different countries between 6 to 24 months (Figures 12 and 13).

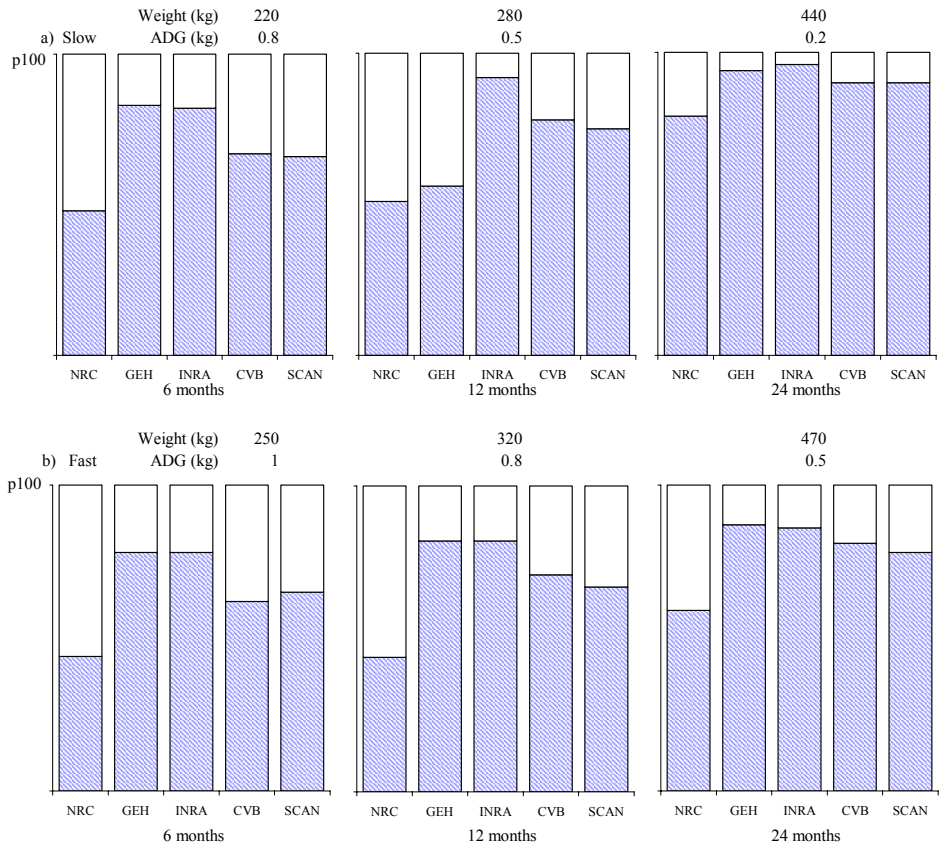


Figure 12. Effect of bodyweight (Weight in kg) and average daily gain (ADG in kg; a) slow and b) fast) on distribution of total energy requirements between Maintenance =  and Growth =  per system (NRC, 1989; GEH, 1994; INRA, 1990; CVB, 1996 and SCAN, 2002).

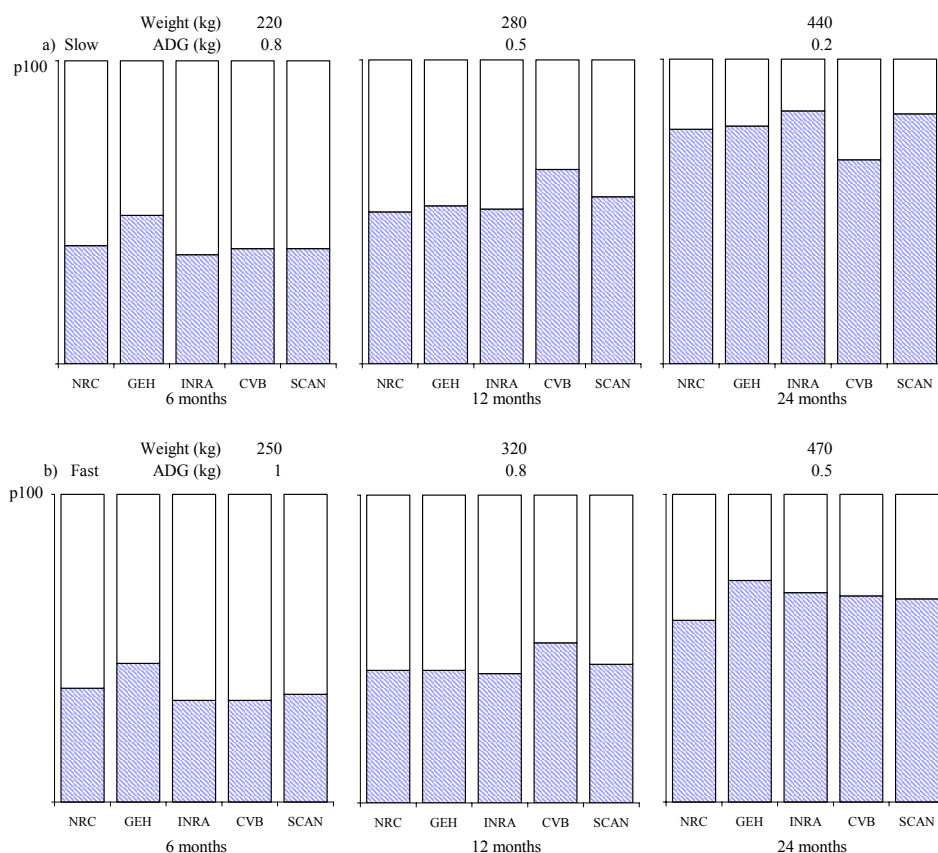


Figure 13. Effect of bodyweight (Weight in kg) and average daily gain (ADG in kg/d; a) slow and b) fast) on distribution of total protein requirements between Maintenance =  and Growth =  per system (NRC, 1989; GEH, 1994; INRA, 1990; CVB, 1996 and SCAN, 2002).

For energy and protein, the proportion of maintenance requirements rises in this interval whatever the estimations. Comparing NRC to other estimations, the proportion of energy requirements for maintenance is always much lower. In all the European estimations the maintenance requirements are estimated using metabolic body weight:  $BW^{0.75}$  whereas body weight is used in NRC estimation. In addition growth requirements are estimated in all the European countries using the variation in the body composition whereas NRC and Scandinavia are using the age of the animals as a criteria in the model. Age is only an indirect thus partial criteria of the variation in body composition as far as it includes probably more or less the variation in maintenance requirements too. It is quite clear when Scandinavian estimations are concerned: growth requirements are calculated using a model where age is the criteria too but maintenance requirements are estimated using  $BW^{0.75}$  simultaneously. As a result the ratio maintenance/growth in total requirements are intermediate between NRC and the other European estimations namely in the early ages. The trend is similar for protein requirements but more significant in the yearling and in the 2 years old than in the weanling.

Of course the proportion of maintenance requirements decrease whatever the systems as far as the ADG increase at the same BW. But the reduction is higher for energy in NRC and to a lesser extent in Scandinavian and CVB estimations than in other ones whatever the age and mainly in 2 years old (Figures 12 and 13). The trend is less pronounced and consistent for nitrogen requirements (Figure 13a and b).

### **Future possibilities for improving current energy and protein requirements and allowances for growing horses within the EU context**

Comparing the recommendations in the different countries, BW can be significantly different (5 to 10p100) according to the age (Tables 9 to 14). The effect is probably limited as far as daily nutrients for maintenance are calculated using models with  $BW^{0.75}$ : +5 to 20p100 comparing 400kg vs 300kg BW for energy and 0 to 5p100 for protein in European estimations. The effect is much higher: +29 and + 33p100 for energy and protein in American estimations where models are using BW. It could contribute to some inconsistency between the different recommendations as far as BW could be different for the same breed between the different country. This was also highlighted by Ellis and Van Tilburg (2002). Body weight curves have to be established for all the main breeds using data obtained in feeding experiments planned for that purpose or/and data obtained in the national studs where the young horses are tested for selection planning.

ADG designed in the different countries are close except for optimal growth suggested in France which is higher. As a result the estimation of daily nutrients should be consistent as far as the models are using ADG. And global method should be preferred as far as efficiency rates of energy and protein for growth have not been yet determined in young horses, or at least implemented to validate factorial calculation. It is well known from beef cattle that proportion of protein which are synthesised could vary widely between breeds or/and sexes as far as the efficiency of energy for protein accretion is much lower than for lipogenesis. Similar differences due to breeds or/and sex effects were determined in heavy breeds by INRA (Agabriel *et al.*, 1984) as far these animals were slaughtered then anatomically dissected (Martin-Rosset *et al.*, 1983b). Such differences are included in the models performed by INRA for estimating energy and protein requirements of males and females in heavy breeds:

- for males:  $UFC/kg/day = 0.0456 BW^{0.75} + 0.0270 G^{1.4}$
- for females:  $UFC/kg/day = 0.0578 BW^{0.75} + 0.0171 G^{1.4}$

Body weight curves should be performed for the main different breeds and sexes.

Body weight curves and nutrients intake should be determined for different breeds and sexes in long term feeding experiments designed for that purpose as far as the slaughter of horses of light breeds is not possible, for estimating accurately the requirements and then the recommended allowances. INRA has obtained in France such data for light breeds: sport breeds (Selle-Français and Anglo-Arab) and race breeds (Thoroughbred and Standardbreds); Ellis and Van Tilburg (2002) have collected similar data from Dutch Warmblood horses in the Netherlands; similar figures can be drawn from experiments conducted with Danish Warmblood horses at DIAS in Denmark; Universities of Molise and Turin in Italy have collected such data in thoroughbred. There are probably other data available in other countries. All those data could be used according to the same methodology for testing the existing models and then may be utilised to develop new European models including the effects of breeds and sexes.

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# Rations for intensive growth in young horses

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## Abstract

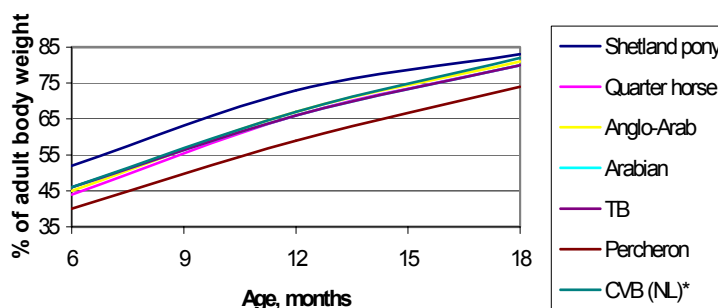
Intensive growth is a widely used expression describing a growth pattern according to a smooth growth curve with no periods of reduced growth or compensatory growth. According to this description, “optimal growth” would probably be a better term. Growth that includes extra fattening of the young animal (prepping for yearling sales) is outside the scope of this paper.

## Introduction

When calculating rations for growing horses, information is needed about the nutrient requirements based on growth parameters, the nutrient content of available feeds and the expected feed intake. In addition, factors concerning climatic conditions and level of activity/exercise have to be considered.

## Description of growth

Growth curves have been described for different breeds of horses by several authors. When expressed as percentage of adult body weight, the differences are remarkably small between different types of horses if we exclude the ponies (Shetland) and heavy horses (Percheron).



\* CVB is not a breed, figures are from the Dutch standard growth curve for horses.

Figure 1. Growth in horses (percent of adult body weight). From Hintz et al. (1997), Hintz (1980) and CVB (1996).

Genetic factors may partly explain why the ponies grow faster and the heavy horses more slowly compared to the other breeds. However, to evaluate figures describing growth, information about the body condition should be available in addition to body weight. Traditions among breeders could otherwise have an influence, as ponies are often fed to achieve higher body condition scores, and the heavy horses are frequently fed to grow slowly during winter and compensate on summer pastures.

As the Dutch growth curve (CVB, 1996) seems to represent the light breeds quite well, this curve can be used for calculating body weight and average daily gain of growing horses.

## Feed intake

Feed intake, as % of BW, varies according to age and the quality and type of feed offered. Housing and climatic conditions may also influence feed intake. Table 1 presents expected feed consumption by young horses.

*Table 1. Expected feed consumption by growing horses (% of BW)\*.*

| Age       | % of body weight |             |          | % of diet |             | Ref.         |
|-----------|------------------|-------------|----------|-----------|-------------|--------------|
|           | Forage           | Concentrate | Total    | Forage    | Concentrate |              |
| 6 months  | 0.5-1.0          | 1.5-3.0     | 2.0-3.5  |           |             | NRC (1989)   |
| 12 months | 1.0-1.5          | 1.0-2.0     | 2.0-3.0  |           |             | NRC (1989)   |
| 18 months | 1.0-1.5          | 1.0-1.5     | 2.0-2.5  |           |             | NRC (1989)   |
| 24 months | 1.0-1.5          | 1.0-1.5     | 1.75-2.5 |           |             | NRC (1989)   |
| 6 months  | 0.5-1.8          | 1.0-3.0     | 2.8-3.5  | 30-65     | 35-70       | Pagan (1998) |
| 12 months | 1.0-2.5          | 0.5-2.0     | 3.0      | 33-80     | 20-66       | Pagan (1998) |

\* As air dry feed

As a rough guideline, feed intake can be estimated to be 3.5-3.0 % of BW for weanlings (6 months) and about 3.0 and 2.5 % of BW for yearlings and two-year old, respectively.

## Ratio concentrate/roughage

The ratio concentrate/roughage in rations for Thoroughbreds varies according to Pagan (1998) from 70/30 to 35/65 for 6 months old, and from 66/33 to 20/80 for yearlings. In a British survey (Harris *et al.*, 1999), typical Thoroughbred rations had a concentrate/roughage ratio of 60/40 for 6 months old, and 50/50 for yearlings. Dry matter intakes were 2.5% and 2.0% of BW for 6 months old and yearlings, respectively. Warm-blooded horses normally get rations with a lower concentrate/roughage ratio than Thoroughbreds (Clausen, E., personal communication).

## Calculation of energy requirement

A comparison between American (NRC, 1989), Dutch (CVB, 1996) and French (Vermorel and Martin-Rosset, 1993) requirements for energy shows that the NRC requirements are 11 – 17% higher compared to CVB when the same growth parameters are being used. The French (INRA) equations seem to overestimate requirements for horses older than 12 months (Table 2).

*Table 2. Energy requirements for growing horses according to NRC, CVB and INRA systems. Adult body weight of horse is 550 kg.*

| Age | ADG, kg <sup>1</sup> | BW, kg <sup>1</sup> | NRC     |                  | CVB              | INRA |
|-----|----------------------|---------------------|---------|------------------|------------------|------|
|     |                      |                     | MJ (DE) | FUh <sup>2</sup> | FUh <sup>3</sup> | UFC  |
| 6 m | 0.83                 | 253                 | 74.7    | 4.9              | 4.4              | 4.7  |
| 12  | 0.51                 | 372                 | 85.0    | 5.6              | 4.8              | 5.7  |
| 18  | 0.31                 | 446                 | 83.7    | 5.5              | 4.8              | 6.2  |
| 24  | 0.19                 | 491                 | 82.9    | 5.5              | 4.8              | 6.5  |

<sup>1</sup> Growth curve from CVB (1995).

<sup>2</sup> Calculating from DE to NE using factor 0.62.

<sup>3</sup> FUh=UFC

In the following examples of rations, energy requirements for growing horses are calculated according to the Dutch requirements (DVB, 1996), which again are based on German requirements (DLG, 1982; Meyer, 1992). To adjust for intensive growth, energy requirements are adjusted to 25% above the calculated values. This also takes into account that young horses increase voluntary activity when fed extra energy.

## Nutrient content of feeds

The quality of roughage varies greatly and must be considered, as well as the recommended use of the compound feeds used. A comparison between “typical” hay qualities in Norway and the UK is shown in Table 3.

*Table 3. Comparison between average nutrient content of meadow hays referred to as “Good”, “Medium” and “Poor” quality in Norway and in the UK (per kg DM).*

| Country | Quality | DE<br>MJ | ME<br>MJ | NE<br>Fuh | CP<br>g | DCP<br>g | Lysine<br>g | Ca<br>g | P<br>g |
|---------|---------|----------|----------|-----------|---------|----------|-------------|---------|--------|
| N       | Good    | 12.2     | 10.3     | 0.77      | 120     | 77.5     | 4.2         | 3       | 2.7    |
| N       | Medium  | 10.5     | 8.8      | 0.64      | 101     | 59.5     | 3.2         | 3       | 2.5    |
| N       | Poor    | 8.9      | 7.5      | 0.54      | 88      | 48       | 2.6         | 3       | 2.5    |
| UK      | Good    | 10.7     | 9        | 0.67      | 120     | 77       | 4.5         | 5.4     | 2.7    |
| UK      | Medium  | 9.5      | 8        | 0.57      | 107     | 66       | 3.8         | 4.8     | 2.5    |
| UK      | Poor    | 7.7      | 6.5      | 0.45      | 80      | 40       | 2.4         | 3       | 2.3    |

Values taken from Norwegian and UK versions of PC-Horse computer programme.

The typical values for Norwegian hay can also be used for hay in the other Nordic countries. The energy content is higher and the calcium content lower in the Norwegian hays compared to the hays from the UK. Very little information is available about what hay qualities are actually being used by the horse breeders. In fact, the majority of breeders do not even have an analysis of the hay used.

Most compound feeds for growing horses (before training is introduced) should not be fed together with other grains, but be given as the only concentrate in addition to roughage. This is to give the total ration an optimum balance of nutrients. However, in most countries it is a common practice for horse breeders to offer grains in addition to the compound feed, and

thereby diluting the concentrations of essential amino acids and some of the minerals and vitamins. Another common practice is to feed supplements (minerals/vitamins) in addition to the compound feed without having identified any nutritional need for the supplement. Table 4 gives examples of nutrient content of compound feeds for growing horses (0.6 to 2 years old). These products are from the UK, Norway and Sweden.

*Table 4. Nutrient content in examples of compound feeds for growing horses from different countries (per kg DM).*

| Country | DE, MJ | ME, MJ | NE, FUh | CP, g | DCP, g | Lysine, g | Ca, g | P, g |
|---------|--------|--------|---------|-------|--------|-----------|-------|------|
| Norway  | 14.4   | 12.9   | 1.1     | 201   | 165    | 10.5      | 18.6  | 9.3  |
| UK      | 14     | 12.6   | 1.1     | 185   | 150    | 8.7       | 15    | 7    |
| Sweden  | 11.5   | 10.3   | 0.9     | 136   | 104    | 6.2       | 6.9   | 5.8  |

There are only small differences in the nutrient composition of the Norwegian and UK compound feeds. The Swedish product has been chosen to give an example of a compound feed with a reduced nutrient content caused by increased inclusion of fibre.

## Rations

In most countries Thoroughbreds are fed more concentrates and less roughage compared to warmblooded riding horses. For the warmbloods, the tradition is to feed roughage ad lib. or semi ad lib. and supply either a balanced compound feed or grains + balancer at 0.4 – 0.6% of expected adult BW (E. Clausen, personal communication). In an experiment with Danish Warmblood (Staun *et al.*, 1995), average daily feed consumption (grass silage and hay) during first winter period was equivalent to 5.4 kg of hay. In addition the horses were given 3.0 kg of compound feed and straw for bedding. The energy content of this ration was calculated to 6.0 FUh., which must be regarded as intensive feeding.

*Table 5. Rations for weanlings (6 months) and yearlings according to the compound feed manufacturers recommendations (550 kg adult BW).*

| Country             | Age  | BW<br>kg | Hay<br>kg | Conc.<br>kg | Total ration<br>kg | Conc.<br>% | Hay<br>% | Ration<br>% of BW |
|---------------------|------|----------|-----------|-------------|--------------------|------------|----------|-------------------|
| Norway              | 6 m  | 253      | 5.5       | 2.8         | 8.3                | 33.7       | 66.3     | 3.3               |
| Norway              | 12 m | 372      | 7         | 2.5         | 9.5                | 26.3       | 73.7     | 2.6               |
| UK                  | 6 m  | 253      | 2.8       | 4.2         | 7                  | 60.3       | 39.7     | 2.8               |
| UK                  | 12 m | 372      | 4.1       | 4.1         | 8.2                | 50.0       | 50.0     | 2.2               |
| Sweden <sup>1</sup> | 6 m  | 253      | 5         | 3           | 8                  | 37.5       | 62.5     | 3.2               |
| Sweden              | 12 m | 372      | 5.5       | 4           | 9.5                | 42.1       | 57.9     | 2.6               |

Air dry feed.

<sup>1</sup> Ad lib. roughage recommended from manufacturer.

Medium hay qualities from Table 3, compound feeds from Table 4 and rations from Table 5 are being used to calculate nutrient content in the following examples of rations.

**Example 1. Ration for weanling (6 months), Norway.**

|               |                |        |           |       |           |           |       |      |
|---------------|----------------|--------|-----------|-------|-----------|-----------|-------|------|
| Age           | Adult BW       |        | BW        |       | ADG       |           |       |      |
| 6 months      | 550 kg         |        | 253 kg    |       | 0.83 kg/d |           |       |      |
| Feed          | DE, MJ         | ME, MJ | NE, Fuh   | CP, g | DCP, g    | Lysine, g | Ca, g | P, g |
| Hay, medium   | 10.5           | 8.8    | 0.64      | 101   | 59.5      | 3.2       | 3     | 2.5  |
| Compound feed | 14.4           | 12.9   | 1.1       | 201   | 165       | 10.5      | 18.6  | 9.3  |
| Ration:       | Hay:           |        | 5.5 kg    |       |           |           |       |      |
|               | Compound feed: |        | 2.8 kg    |       |           |           |       |      |
|               | Requirement    |        | In ration |       | Balance   |           | %     |      |
| FUh           | 5.5            |        | 5.6       |       | 0.06      |           | 101   |      |
| DCP, g        | 704.3          |        | 672.6     |       | -31.7     |           | 95    |      |
| Lysine, g     | 38.0           |        | 40.1      |       | 2.0       |           | 105   |      |
| Ca, g         | 45.9           |        | 58.6      |       | 12.7      |           | 128   |      |
| P, g          | 25.4           |        | 34.0      |       | 8.5       |           | 134   |      |

Ca/P: 1.7

DCP/FUh: 121 g

Total intake (air dry feed): 3.3% of BW

**Example 2. Ration for yearling, Norway.**

|               |                |        |           |       |           |           |       |      |
|---------------|----------------|--------|-----------|-------|-----------|-----------|-------|------|
| Age           | Adult BW       |        | BW        |       | ADG       |           |       |      |
| 12 months     | 550 kg         |        | 372       |       | 0.51 kg/d |           |       |      |
| Feed          | DE, MJ         | ME, MJ | NE, Fuh   | CP, g | DCP, g    | Lysine, g | Ca, g | P, g |
| Hay, medium   | 10.5           | 8.8    | 0.64      | 101   | 59.5      | 3.2       | 3     | 2.5  |
| Compound feed | 14.4           | 12.9   | 1.1       | 201   | 165       | 10.5      | 18.6  | 9.3  |
| Ration:       | Hay:           |        | 7.0 kg    |       |           |           |       |      |
|               | Compound feed: |        | 2.5 kg    |       |           |           |       |      |
|               | Requirement    |        | In ration |       | Balance   |           | %     |      |
| FUh           | 5.9            |        | 6.1       |       | 0.2       |           | 103   |      |
| DCP, g        | 559.4          |        | 705.0     |       | 145.65    |           | 126   |      |
| Lysine, g     | 30.2           |        | 41.4      |       | 11.2      |           | 137   |      |
| Ca, g         | 39.0           |        | 57.5      |       | 18.5      |           | 147   |      |
| P, g          | 21.6           |        | 34.7      |       | 13.1      |           | 161   |      |

Ca/P: 1.7

DCP/FUh: 116 g

Total intake (air dry feed): 2.7% of BW

**Example 3. Ration for weanling (6 months), UK.**

|               |                |        |           |       |           |           |       |      |
|---------------|----------------|--------|-----------|-------|-----------|-----------|-------|------|
| Age           | Adult BW       |        | BW        |       | ADG       |           |       |      |
| 6 months      | 550 kg         |        | 253 kg    |       | 0.83 kg/d |           |       |      |
| Feed          | DE, MJ         | ME, MJ | NE, Fuh   | CP, g | DCP, g    | Lysine, g | Ca, g | P, g |
| Hay, medium   | 9.5            | 8      | 0.57      | 107   | 66        | 3.8       | 4.8   | 2.5  |
| Compound feed | 14             | 12.6   | 1.1       | 185   | 150       | 8.7       | 15    | 7    |
| Ration:       | Hay:           |        | 2.8kg     |       |           |           |       |      |
|               | Compound feed: |        | 4.2 kg    |       |           |           |       |      |
|               | Requirement    |        | In ration |       | Balance   |           | %     |      |
| FUh           | 5.51           |        | 5.15      |       | -0.36     |           | 93    |      |
| DCP, g        | 704.3          |        | 700.05    |       | -4.25     |           | 99    |      |
| Lysine, g     | 38.03          |        | 44.66     |       | 6.63      |           | 117   |      |
| Ca, g         | 45.85          |        | 61.6      |       | 15.75     |           | 134   |      |
| P, g          | 25.42          |        | 31.08     |       | 5.65      |           | 122   |      |

Ca/P: 2.0

DCP/FUh: 136 g

Total intake (air dry feed): 2.8% of BW

**Example 4. Ration for yearling, UK.**

|               |                |        |           |       |           |           |       |      |
|---------------|----------------|--------|-----------|-------|-----------|-----------|-------|------|
| Age           | Adult BW       |        | BW        |       | ADG       |           |       |      |
| 12 months     | 550 kg         |        | 372       |       | 0.51 kg/d |           |       |      |
| Feed          | DE, MJ         | ME, MJ | NE, Fuh   | CP, g | DCP, g    | Lysine, g | Ca, g | P, g |
| Hay, medium   | 9.5            | 8      | 0.57      | 107   | 66        | 3.8       | 4.8   | 2.5  |
| Compound feed | 14             | 12.6   | 1.1       | 185   | 150       | 8.7       | 15    | 7    |
| Ration:       | Hay:           |        | 4.1 kg    |       |           |           |       |      |
|               | Compound feed: |        | 4.1 kg    |       |           |           |       |      |
|               | Requirement    |        | In ration |       | Balance   |           | %     |      |
| FUh           | 5.9            |        | 5.7       |       | -0.3      |           | 96    |      |
| DCP, g        | 559.4          |        | 758.7     |       | 199.3     |           | 136   |      |
| Lysine, g     | 30.2           |        | 43.9      |       | 13.7      |           | 145   |      |
| Ca, g         | 39.0           |        | 69.7      |       | 30.7      |           | 179   |      |
| P, g          | 21.6           |        | 33.2      |       | 11.6      |           | 154   |      |

Ratio Ca/P: 2.1

DCP/FUh: 133 g

Total intake (air dry feed): 2.2% of BW



**Example 5. Ration for weanling (6 months), Sweden.**

|               |                |        |           |       |           |           |       |      |
|---------------|----------------|--------|-----------|-------|-----------|-----------|-------|------|
| Age           | Adult BW       |        | BW        |       | ADG       |           |       |      |
| 6 months      | 550 kg         |        | 253 kg    |       | 0.83 kg/d |           |       |      |
| Feed          | DE, MJ         | ME, MJ | NE, Fuh   | CP, g | DCP, g    | Lysine, g | Ca, g | P, g |
| Hay, medium   | 10.5           | 8.8    | 0.64      | 101   | 59.5      | 3.2       | 3     | 2.5  |
| Compound feed | 11.5           | 10.3   | 0.9       | 136   | 104       | 6.2       | 6.9   | 5.8  |
| Ration:       | Hay:           |        | 5.0kg     |       |           |           |       |      |
|               | Compound feed: |        | 3.0 kg    |       |           |           |       |      |
|               | Requirement    |        | In ration |       | Balance   |           | %     |      |
| FUh           | 5.51           |        | 4.96      |       | -0.55     |           | 90    |      |
| DCP, g        | 704.3          |        | 520       |       | -184.3    |           | 74    |      |
| Lysine, g     | 38.03          |        | 29.7      |       | -8.33     |           | 78    |      |
| Ca, g         | 45.85          |        | 30.5      |       | -15.35    |           | 67    |      |
| P, g          | 25.42          |        | 25.5      |       | 0.08      |           | 100   |      |

Ca/P: 1.2

DCP/FUh: 105 g

Total intake (air dry feed): 3.2% of BW

**Example 6. Ration for yearling, Sweden.**

|               |                |        |           |       |           |           |       |      |
|---------------|----------------|--------|-----------|-------|-----------|-----------|-------|------|
| Age           | Adult BW       |        | BW        |       | ADG       |           |       |      |
| 12 months     | 550 kg         |        | 372       |       | 0.51 kg/d |           |       |      |
| Feed          | DE, MJ         | ME, MJ | NE, Fuh   | CP, g | DCP, g    | Lysine, g | Ca, g | P, g |
| Hay, medium   | 10.5           | 8.8    | 0.64      | 101   | 59.5      | 3.2       | 3     | 2.5  |
| Compound feed | 11.5           | 10.3   | 0.9       | 136   | 104       | 6.2       | 6.9   | 5.8  |
| Ration:       | Hay:           |        | 5.5 kg    |       |           |           |       |      |
|               | Compound feed: |        | 4.0 kg    |       |           |           |       |      |
|               | Requirement    |        | In ration |       | Balance   |           | %     |      |
| FUh           | 5.94           |        | 5.98      |       | 0.04      |           | 101   |      |
| DCP, g        | 559.38         |        | 635       |       | 75.62     |           | 114   |      |
| Lysine, g     | 30.21          |        | 36.45     |       | 6.24      |           | 121   |      |
| Ca, g         | 39             |        | 37.75     |       | -1.25     |           | 97    |      |
| P, g          | 21.58          |        | 31.55     |       | 9.97      |           | 146   |      |

Ratio Ca/P: 1.2

DCP/FUh: 106 g

Total intake (air dry feed): 2.6% of BW

Rations show that there is marginal energy supply to weanlings in the examples from the UK and Sweden. In the example from the UK, total ration is 2.8% of BW and it could be expected that weanlings should be able to consume more than 2.8 kg of hay/d. In the example from Sweden, the low energy content of the compound feed could represent a problem as the total intake is 3.2% of BW.

Protein supply is marginal for weanlings in the Norwegian ration and low (also for lysine) in the Swedish ration.

Calcium and Phosphorous supply for the weanlings is about 20-30% above requirements in the Norwegian and UK rations, but low in the Swedish ration.

Rations for yearlings seem to meet requirements for energy and protein in all countries, and amounts of calcium and phosphorous are adequate or above requirements.

These examples illustrate that the most critical period is before the horses are 12 months of age. In addition to a well-balanced compound feed (or grains + balancer), high quality roughage is crucial for the young horses.

## Micro minerals and vitamins

In addition to supply of energy, protein and macro-minerals, a good compound feed (or balancer) should balance micro-minerals and vitamins in total rations.

Figures 2-4 show total ration content of micro-minerals and vitamins for the examples of rations for weanlings. All rations have adequate or higher contents of micro-minerals and vitamins. The common practice of supplying extra minerals and vitamins, regardless of nutritional status, should therefore be avoided when a good compound feed or grain balancer is used in accordance with the manufacturers' recommendations.

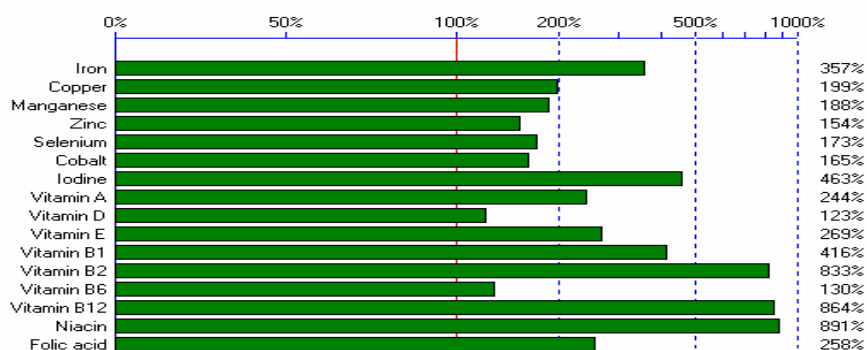


Figure 2. Micro minerals and vitamins in diet for weanling (6 months), Norway.

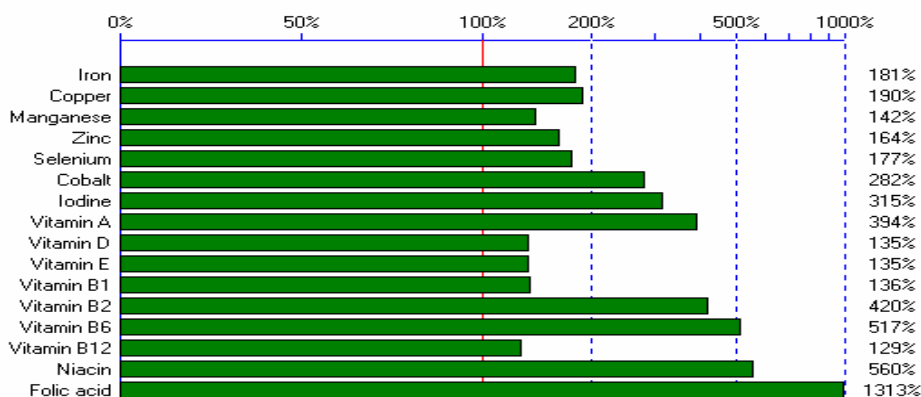


Figure 3. Micro minerals and vitamins in diet for weanling (6 months), UK.

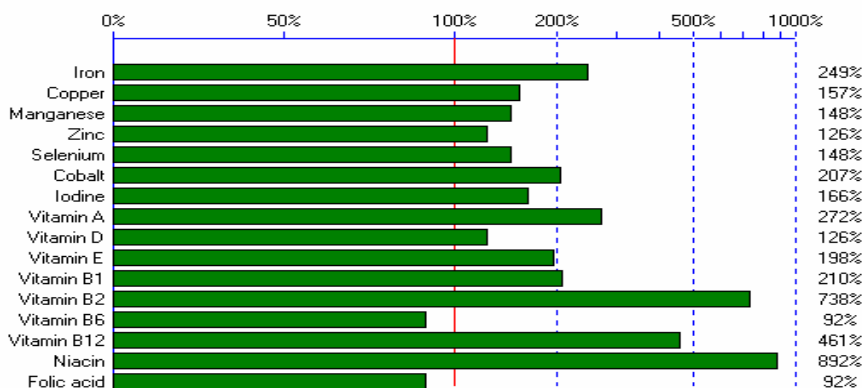


Figure 4. Micro minerals and vitamins in diet for weanling (6 months), Sweden.

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## Feeding the young horse managed with moderate growth

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### Abstract

Young horses devoted to leisure, as well as most of sport horses are not broken in until 42 months of age. Their growth rate can be temporary limited during winter periods in order to implement their capabilities of compensatory growth during the grazing periods, and hence to maximise the use of forage based diets.

Nevertheless, such a discontinuous growth pattern can lead to the same body weight and size between 42 to 48 months of age even if growth is limited during all the subsequent winter periods.

Forages fed during these periods, can be hay fed alone or associated with straw, or preserved forages such as: grass silage, wrapped silage or maize silage.

Nutrients contents and ingestibility of such forages, as well as feed intake of young horses at 1, 2 and 3 years of age have been evaluated, as well as animal's performances (BW, Size) according to diet composition (ratio concentrate/forages) through experimental trials carried out by the French National studs and INRA research centre since 1970.

*Keywords: Horse sport leisure, discontinuous growth, forage*

### Introduction

In contrast with race horses, such as French Trotters or thoroughbreds, whose career starts at 2 years of age (entering training programme at 18 months of age), most of the saddle horses (especially leisure horses) are not broken in until 42 months of age. The growth of such horses does not need to be sustained during the two first winter periods, because of horse capabilities to have a compensatory growth, during summer periods at pasture, depending on the level and the duration of the winter feed restriction (Martin-Rosset *et al.*, 1984; Trillaud-Geyl *et al.*, 1990).

The growth limitation during winter allow maximisation of both pasture utilisation during summer periods and forage utilisation during winter, reducing concentrate use in the diets (Trillaud-Geyl *et al.*, 1986; Bigot *et al.*, 1987). The young horse can enhance its voluntary intake when fed *ad libitum* to compensate, at least on DM based intake, the restriction of the amount of concentrate fed (Martin-Rosset and Doreau, 1984). Forage fed during winter period may be either dry (hay, straw) or preserved (grass silage or maize silage). The condition for using the latter have been well defined for horses (Martin-Rosset and Doreau, 1984; Bigot *et al.*, 1987; Trillaud-Geyl and Martin-Rosset, 2004).

The percentage of concentrate in the diet varies from 10 to 20p100 of total DM intake, depending on the age of the animal and the type of forage fed. These feeding practices are based on several feeding experiments conducted since the seventies by the French National

Studs and INRA. All these results contributed to define feeding systems in accordance with husbandry systems (INRA, 1990, Micol and Martin-Rosset, 1995).

## Principle and baseline of ration formulation

Formulation of rations means:

- 1 choosing available feeds according to expected growth rate and type of horse breed;
- 2 define the amount of the chosen feeds in order to offer the amount of nutrients or feeds requested to meet the animals' requirements corresponding to the objectives of production and growth.

The calculation of nutrients requirements and intake rely on information on:

- 1 Animal characteristics: age, BW, growth rate to determine the requirements, whereas the DM intake (expressed as Kg DM/100 kg of BW, or g DM/Kg of BW<sup>0.75</sup>) allows recommended allowance calculation. All these information is stated in the INRA tables for recommended allowance (1990). But they can be calculated too using the nutritional equations defined by INRA (Martin-Rosset *et al.*, 1994).
- 2 The feeds characteristics: ingestibility of forage expressed in Kg DM/100kg of BW, the nutritional values of feeds (UFC, MADC, ...), experimentally measured, and provide in the INRA tables of feeds composition and nutritive values (INRA, 1990).

Finally, the intake depends also on substitution rate between forages and concentrates. It was measured for the main types of forages to determine the optimal composition of rations fed to horses (Martin-Rosset and Doreau, 1984, Trillaud-Geyl and Martin-Rosset, 2004). It is stated in the INRA tables of recommended allowance by high or low DM intake levels for different ages (Table 1).

*Table 1. Recommended nutrient allowances for saddle horses (average mature body weight: 550 kg),(INRA 1990).*

| Age<br>(months) | Average live<br>BW over the<br>period (kg) | Growth rate |              | Daily nutrients intake |             |           |          |           |           | DM<br>intake<br>(kg) |
|-----------------|--------------------------------------------|-------------|--------------|------------------------|-------------|-----------|----------|-----------|-----------|----------------------|
|                 |                                            | Level       | ADG<br>(g/d) | UFC                    | MADC<br>(g) | Ca<br>(g) | P<br>(g) | Mg<br>(g) | Na<br>(g) |                      |
| 8 - 12          | 340                                        | optimal     | 750-850      | 5.8                    | 630         | 41        | 23       | 10        | 13        | 6.0-8.5              |
|                 | 300                                        | moderate    | 450-550      | 4.8                    | 470         | 30        | 18       | 10        | 10        | 5.5-8.0              |
| 20 - 24         | 500                                        | optimal     | 450-550      | 7.3                    | 450         | 39        | 22       | 10        | 14        | 8.0-11.0             |
|                 | 470                                        | moderate    | 200-300      | 6.4                    | 360         | 31        | 18       | 10        | 13        | 7.5-10.0             |
| 30 - 36         | 530                                        | optimal     | 200-300      | 7.0                    | 360         | 34        | 19       | 10        | 14        | 8.5-12.0             |
|                 | 510                                        | moderate    | 0-100        | 6.4                    | 290         | 27        | 16       | 10        | 13        | 9.0-11.0             |

Formulation of rations can be performed either by:

- 1 using the graphical method, that can determine optimal diet from the graphical representation of feeds potentially available for the horses represented on the graph using their coordinates: UFC/Kg DM value as abscissa, their MADC/kg DM value as ordinate;
- 2 using the computing method, that is implementing CHEVALRATION file based on the graphical method and the recommended allowances edited by INRA 1990.

All these methods have been established for the young growing horse on data drawn from long term feeding experiments conducted by the French National Studs and INRA.

## Diets and rations

The growing horse can be fed during 3 winter post weaning periods from 8 months of age until the breaking in period, with dry (hay, straw) or preserved (silage, haylage) forages. In all the studies reported here, the young Anglo-Arab and French saddle horses weighted an average BW of: 260 to 270kg at 8 months of age, 330 to 340kg at 1 year of age, 430 to 450 at 2 years of age and 520 to 530kg at 3 years of age.

### Diets based on dry forages

The forage fed can be a first cut meadow hay of average quality: containing 34% of CF an 9% of CP, i.e. 0.55 UFC and 45g of MADC/kg DM in average for a hay harvested at the stage of full earing/beginning of blossoming according to the INRA tables (1990).

The hay is fed *ad libitum* and supplemented with 1kg of a concentrate feed.

Limited amounts of meadow hay can be fed associated with straw supplemented with 1kg of concentrate feed. The hay is a second cut hay of average quality, containing 31% of CF and 13% of CP, i.e. 0.60 UFC and 65g of MADC/kg DM.

#### *During the first winter period: 6-12 months*

The total amount of DM intake is of 2.5 to 2.6 kg/100 kg of BW with a hay based diet and the concentrate feed represents 12p100 of the ration. The average daily gain permitted is 400g/day (Table 2).

#### *During the second winter period: 18-24 months*

The animals are fed a mixed diet: limited amount of first cut meadow hay and wheat straw *ad libitum*. The total amount of DM intake is of 2.0 to 2.4 kg/100 kg of BW. The straw and the concentrate feed represent 31p100 and 10p100 of the total ration respectively. The average daily gain is 100g/day (Table 2).

#### *During the third winter period: 30-36 months*

The animals are fed the same diet as the 2 years old. The total amount of DM intake is of 2.2 to 2.4 kg/100 kg of BW. The straw and the concentrate feed represent 25p100 and 10p100 of the total ration respectively. The average daily gain is 20g/day (Table 2).

### Diets based on grass silage

The forage fed is a prewilted grass silage that leads to a minimum of 22-25% of DM content. It can be either meadow grass or a mixture of ryegrass and cocksfoot grass cut at a stage of full earing: containing 31-32% of CF an 10% of CP, i.e. 0.60-0.65 UFC and 50-60 g of MADC/kg DM in average. The preservation quality is according to the INRA table (1981): "average" to "good" (Table 3).

Table 2. Different type of diets studied by Haras Nationaux and INRA (1970-2002).

| Young saddle horses managed with moderate growth rates |                                        |                                             |                                                                                      |                                       |                                       |                |
|--------------------------------------------------------|----------------------------------------|---------------------------------------------|--------------------------------------------------------------------------------------|---------------------------------------|---------------------------------------|----------------|
| Age                                                    | Diets                                  |                                             | Type of forages and intake level<br>(limited or <i>ad libitum</i> )                  | Daily total<br>DM intake <sup>2</sup> | Daily total<br>DM intake <sup>2</sup> | Growth<br>rate |
|                                                        | Forage<br>(p100<br>TDMI <sup>1</sup> ) | Concentrate<br>(p100<br>TDMI <sup>1</sup> ) |                                                                                      | kg DM/day                             | kg DM/100<br>kg BW <sup>3</sup>       | g / day        |
| 6-12 months                                            | 88                                     | 12                                          | <i>Ad libitum</i> Meadow hay                                                         | 7.5 - 8.0                             | 2.5 - 2.6                             | 400            |
|                                                        | 88                                     | 12                                          | Limited meadow hay + <i>Ad libitum</i><br>straw (28p100)                             | 7.0 - 7.5                             | 2.3 - 2.4                             | 250            |
|                                                        | 83                                     | 17                                          | <i>Ad libitum</i> Pre wilted grass silage<br>(22% DM) limited meadow<br>hay (30p100) | 5.0 - 5.5                             | 1.5 - 1.6                             | 250            |
|                                                        | 84                                     | 16                                          | Wrapped grass silage (48% DM)<br><i>Ad libitum</i>                                   | 7.0 - 7.5                             | 2.3 - 2.5                             | 500            |
|                                                        | 84                                     | 16                                          | Maize silage (30% DM) <i>Ad libitum</i>                                              | 5.0 - 5.2                             | 1.7 - 2.0                             | 680            |
|                                                        |                                        |                                             |                                                                                      |                                       |                                       |                |
| 18-24 months                                           | 90                                     | 10                                          | Limited meadow hay +<br><i>Ad libitum</i> straw (31p100)                             | 8.3 - 9.0                             | 2.0 - 2.2                             | 100            |
|                                                        | 88                                     | 12                                          | <i>Ad libitum</i> Pre wilted grass<br>silage (22% DM)                                | 7.0 - 7.5                             | 1.7 - 1.8                             | 200            |
|                                                        | 86                                     | 14                                          | Wrapped grass silage (60% DM)<br><i>Ad libitum</i>                                   | 10.5 - 11                             | 2.2 - 2.5                             | 390            |
|                                                        | 93                                     | 7                                           | Maize silage (30% DM) <i>Ad libitum</i>                                              | 6.0 - 8.4                             | 1.5 - 2.0                             | 310            |
|                                                        |                                        |                                             |                                                                                      |                                       |                                       |                |
| 30-36 months                                           | 90                                     | 10                                          | Limited meadow hay (65p100)<br>+ <i>Ad libitum</i> straw (25p100)                    | 10.0 - 12.0                           | 2.2 - 2.4                             | 20             |
|                                                        | 87                                     | 13                                          | <i>Ad libitum</i> Pre wilted grass silage<br>(22% DM)                                | 7.5 - 8.0                             | 1.5 - 1.6                             | 20             |
|                                                        | 86                                     | 14                                          | Wrapped grass silage (55% DM)<br><i>Ad libitum</i>                                   | 11.5 - 12.0                           | 2.4 - 2.5                             | 50             |
|                                                        | 89                                     | 11                                          | Maize silage (30% DM) <i>Ad libitum</i>                                              | 7.5 - 8.2                             | 1.5 - 1.6                             | 280            |
|                                                        |                                        |                                             |                                                                                      |                                       |                                       |                |

<sup>1</sup> TDMI = Total Dry Matter Intake

<sup>2</sup> DM = Dry Matter

<sup>3</sup> BW = Body Weight

#### *During the first winter period: 6-12 months*

The total amount of DM intake is low: 1.5 to 1.6 kg/100 kg of BW despite the introduction of hay in the diet. The hay and the concentrate feed represent 30p100 and 17p100 of the total ration respectively. The average daily gain is low 250g/day (Table 2).

#### *During the second winter period: 18-24 months*

The total amount of DM intake remain low: 1.7 to 1.8 kg/100 kg of BW. Introduction of hay is not needed. The concentrate feed represent 12p100 of the total ration. The average daily gain is 200g/day (Table 2).

#### *During the third winter period: 30-36 months*

The total amount of DM intake are low and not higher than those measured at 2 years old: 1.5 to 1.6 kg/100 kg of BW. Introduction of hay is not needed. The concentrate feed represent 13p100 of the total ration. The average daily gain permitted are very low 20g/day (Table 2).



Table 3. Tables INRA for evaluating silage's preservation quality (according to Dulphy and Demarquilly, 1981).

| Class     | Volatile fatty acids<br>(mmoles/kg DM) | Acetic acid<br>g / kg DM | Butyric acid<br>g / kg DM | N - NH <sub>3</sub> % total N |         |               | Soluble N<br>% total N |
|-----------|----------------------------------------|--------------------------|---------------------------|-------------------------------|---------|---------------|------------------------|
|           |                                        |                          |                           | Maize                         | Alfalfa | Others plants |                        |
| Excellent | < 330                                  | < 20                     | 0                         | < 5                           | < 8     | < 7           | < 50                   |
| Average   | 330-660                                | 20-40                    | < 5                       | 5-10                          | 8-12    | 7-10          | 50-60                  |
| Good      | 660-1000                               | 40-55                    | > 5                       | 10-15                         | 12-15   | 10-15         | 60-70                  |
| Bad       | 1000-1330                              | 55-75                    | > 5                       | 15-20                         | 16-20   | 15-20         | > 65                   |
| Very bad  | > 1330                                 | > 75                     | > 5                       | > 20                          | > 20    | > 20          | > 75                   |

According to species the following criteria well be preferred :

\* acetic acid and VFA for graminea (namely ryegrass)

\* the proportion of N-NH<sub>3</sub> and VFA for legumes (namely Alfalfa)

The following criteria will be preferred according the priority in the evaluation or/and the correction to be done:

|                      | % DM | pH | N-NH <sub>3</sub><br>% total N | Soluble N | Lactic acid<br>g / kg DM | Volatif fatty<br>acids g / kg DM | Alcohol<br>g / kg DM |
|----------------------|------|----|--------------------------------|-----------|--------------------------|----------------------------------|----------------------|
| Correction for DM    |      |    | NH <sub>3</sub> X              |           | X                        |                                  | X                    |
| Preservation quality |      | X  | X                              |           |                          | X Butyric acid                   |                      |
| Nitrogen value       |      |    | X                              | X         |                          |                                  |                      |
| Ingestibility        | X    |    | X                              | X         |                          | X Acetic acid                    |                      |

### Diets based on wrapped silage

The forage fed is harvested after drying a short time that leads to a DM content of 45 to 65%. It can be either meadow grass or a mixture of ryegrass and cocksfoot grass cut at coming into earing stage: containing 28-30% of CF an 11-12% of CP, ie. 0.65-0.70 UFC and 60-70g of MADC/kg DM in average. The preservation quality is according to the INRA table (1981): "excellent" (Table 3). The wrapped silage is fed *ad libitum*.

#### *During the first winter period: 6-12 months*

The total amount of DM intake is high: 2.3 to 2.5 kg/100 kg of BW. The wrapped silage is harvested at 48% DM. The concentrate feed represents 16p100 of the total ration. The average daily gain is 500g/day (Table 2).

#### *During the second winter period: 18-24 months*

The total amount of DM intake is very high: 2.2 to 2.5 kg/100 kg of BW. The wrapped silage is harvested at 60% of DM. The concentrate feed represents 14p100 of the total ration. The average daily gain is 390g/day (Table 2).

#### *During the third winter period: 30-36 months*

The total amount of DM intake is also high: 2.4 to 2.5 kg/100 kg of BW. The wrapped silage is harvested at 55p100 of DM. The concentrate feed represents 14p100 of the total ration. The average daily gain is 50g/day (Table 2).

## Diets based on maize silage

The maize is harvested at a dough/flint stage with a DM content of 30 to 36% with an average proportion of ears (55 to 65p100). Nutrient content and nutritive value are “good”: containing 20-22% of CF and 7-8% of CP, ie. 0.75-0.80 UFC and 25-30 g of MADC/kg DM in average. The preservation quality is according to the INRA table (1981) “excellent” (Table 3). The maize silage is fed *ad libitum*.

*During the first winter period: 6-12 months*

The total amount of DM intake is rather high: 1.7 to 2.0 kg/100 kg of BW. The concentrate feed represents 16p100 of the total ration. The average daily gain is very high 680g/day (Table 2).

*During the second winter period: 18-24 months*

The total amount of DM intake is still high: 1.5 to 2.0 kg/100 kg of BW. The concentrate feed only represents 7p100 of the total ration. The average daily gain is high 310g/day (Table 2).

*During the third winter period: 30-36 months*

The total amount of DM intake is high: 1.5 to 1.6 kg/100 kg of BW. The concentrate feed represents 11p100 of the total ration. The average daily gain is very high 280g/day (Table 2).

## Practical consequences

The total amount of DM intake varies according to:

- 1 - the animal characteristics: age and body weight;
- 2 - the type and the characteristics of the forages: dry forage, grass or maize silage, their chemical composition and nutritional values, as well as the quality of preservation;
- 3- the proportion of concentrate feed in the ration.

## Characteristics of the forages and conditions of utilisation

Hays fed to yearlings after weaning should be at least of good quality if they are fed *ad libitum*: first cut; e.g. 0.60 UFC and 50 g of MADC/kg of DM according to INRA tables (1990), supplemented with a limited amount of concentrate feed.

When mixed rations are fed, second cut hays should be harvested at the 7 weeks leafy stage: 0.65 UFC-70 g of MADC/kg of DM according to INRA tables (1990) to be fed to one year old. First cut hays harvested at the full earing stage (0.50-0.60 UFC and 45-50 g of MADC/kg DM, tables INRA 1990) are adequate for 2 and 3 years old if they are fed *ad libitum*.

Grass silages harvested at less than 22p100 of DM are not recommended for horses. The intake is very low, even if meadow hay is offered as a supplement. The high ammoniacal nitrogen and volatile fatty acids concentrations (namely acetic acid) added to the undesirable effect of the low DM content lead to chronic digestive disorders. Prewilted silages containing 25 to 30p100 DM are better accepted and ingested. They do not lead to recurrent digestive disorders if they are well preserved.

Wrapped silage, with 45-50p100 DM is probably the best solution. The intake is high thanks to its DM content and good preservation quality, when harvesting and preservation rules have been implemented. They must be fed *ad libitum*. Wrapped silage takes the advantage over hay in that they allow harvesting forage at a earlier stage and thus achieve a higher nutritive value: 0.60-0.75 UFC and 60-70 g of MADC/kg DM depending on whether they are meadow grass, a mixture of ryegrass, cocksfoot or even alfalfa/cocksfoot mixture (INRA tables, 1990).

Maize silage is a very interesting forage because of its high nutritive value. It is highly recommended for horses if the silage is harvested at a minimum of 30% DM content and well preserved. It must be fed *ad libitum* to yearlings and in more or less limited amounts to 2 and 3 years old.

Generally, all silages must be fed to young horses after a 3 to 4 weeks adaptation period to diet after weaning and of 2 weeks for 2 to 3 years old. According to the type of silage, adaptation takes longer for silage (grass or maize) compared to wrapped silage. The DM content and preservation quality may also account in the duration of the adaptation period to the diet. An evaluation of preservation quality must be carried out in a laboratory to determine and control this quality according to the tables of INRA (1981), (Table 3).

### **Animal characteristics: voluntary DM intake**

Voluntary intake of young horses depends on their energy requirements and it is expressed in g DM/kg BW<sup>0.75</sup> and on the development of the gut and it is expressed in kg DM/100kg of BW, which is important with forage based diets.

When forages are fed 83 to 90p100 of the ration to perform moderate growth rates, total amount of DM intake/100kg of BW is as much high as the animal is young.

Intake ranges from 1.7 to 2.6 kg DM/100kg BW with yearlings whereas it varies from 1.5 to 2.2 kg DM/100 kg BW with 3 years old owing to a smaller gut development of the one year old. Indeed the gut development takes place between 12 and 18 months of age during pasture season (Martin-Rosset *et al.*, 1983).

The amount of total DM intake per kg of metabolic BW (BW<sup>0.75</sup>) is all the more higher as the energetic requirements per kg of BW<sup>0.75</sup> are high: 110g, 99g and 98g/kg BW<sup>0.75</sup> at 1, 2 and 3 years of age. The amount of total DM intake also depends on the growth performances particularly with yearlings whose potential growth rate is high.

### **Proportion of concentrate feed in the ration: substitution rate**

The amount of forage ingested closely depends on the amount of concentrate feed offered in the diet. The amount of forage ingested is reduced when the amount of concentrate increases. This reduction is evaluated as the substitution rate which represents the reduction of forage DM intake per kg DM of additional concentrate feed fed.

The substitution rate varies from 0.5 to 0.8; 0.4 to 0.6 per kg DM of concentrate feed for yearlings and 2 to 3 years old horses with hay based diets and maize silage respectively supplemented with low amount of concentrate (10 to 17p100). This substitution is linked to the nutritive value of the forage fed. It is the all the more important as the energetic value of the forage is limited: hay 0.50 to 0.60 UFC/ kg DM, compared to silage maize 0.70 to 0.85

UFC/ kg DM; the wrapped silage having an intermediate position. The substitution rate may be much more important when the proportion of concentrate in the diet rises over 30% of the ration: 1.3 for hay, 0.5 to 0.9 for wrapped silage and 0.6 to 0.8 for maize silage (Martin-Rosset and Doreau, 1984; Trillaud-Geyl and Martin-Rosset, 2004).

### **Characteristics of concentrate feeds**

Compound feeds used for supplementation of forage (dry or silage) based diets are essentially composed of grains (maize or barley), cakes to balance energy and protein contents of the diets.

The proportion of cake in the concentrate feed is as high as the diet is based on prewilted silage because the relatively high amount of soluble nitrogen, which limits the MADC value of this type of forage. Proportion of cakes is relatively higher, too, in the case of diets including straw distribution.

Compound concentrate feeds used to supplement maize silage diets are essentially composed of cakes in order to balance the low MADC value of maize silage. The cakes used for yearling are almost soya because of its excellent amino acid balance. Rapeseed, sunflower, linseed or peanut cakes can be used for 2-3 year old horses. Dehydrated alfalfa, or leguminous seeds (peas, faba beans, lupine seed) can be advantageously used for the young horses. Corn gluten feed is of limited use for the yearling because of its low ingestibility (Trillaud-Geyl, 1995).

Energy (UFC/kg DM) and protein (g MADC/kg DM) concentration of supplement feeds must be higher for 1 year old horses than for 2-3 years old. Whereas the requirements of 2-3 years old horses expressed by  $\text{kg BW}^{0.75}$  decrease, their forage intake capacity increases sufficiently to meet a rising proportion of their nutrients requirements (Table 1).

Comparing 2-3 years old horses to yearling, energy concentration of the diet does not increase: 0.60 UFC/kg DM in all the cases for moderate growth rate, whereas protein concentration decreases with age 59 to 26 g MADC/kg DM for 1 and 3 years old horses respectively.

Diet must be well balanced with major minerals (Ca, P, Mg) oligo-elements, (Cu, Zn, particularly Se) and with vitamin A and D using an appropriated mineral vitamin supplement included into the compound feed or given aside in the case of raw materials issuing from home farm.

The matter is to fit the balances recommended in the table of optimum nutritive concentration of diets proposed by INRA (1990) (Table 4).

Table 4. Optimum nutritive concentration of horse rations (per kg of dry matter intake according to the recommended allowances; INRA, 1990).

|                             |         | Growing     |              |              |
|-----------------------------|---------|-------------|--------------|--------------|
|                             |         | 6-12 months | 18-24 months | 30-36 months |
| Energy                      |         |             |              |              |
| UFC                         |         | 0.65-0.95   | 0.60-0.90    | 0.60-0.80    |
| Nitrogen                    |         |             |              |              |
| MADC                        | (g)     | 55-90       | 40-55        | 30-40        |
| Minerals                    |         |             |              |              |
| Calcium                     | (g)     | 5.5         | 3.8          | 3.3          |
| Phosphor                    | (g)     | 3.0         | 2.2          | 1.9          |
| Magnesium                   | (g)     | 1.6         | 1.1          | 1.1          |
| Sodium                      | (g)     | 1.8         | 1.6          | 1.4          |
| Potassium                   | (g)     | 3.0         | 6.0          | 1.4          |
| Sulphur                     | (g)     | 1.5         | -            | -            |
| Trace elements <sup>1</sup> |         |             |              |              |
| Iron                        | (mg)    | 80-100      | 80-100       | 80-100       |
| Copper                      | (mg)    | 10          | 10           | 10           |
| Zinc                        | (mg)    | 50          | 50           | 50           |
| Manganese                   | (mg)    | 40          | 40           | 40           |
| Cobalt                      | (mg)    | 0.1-0.3     | 0.1-0.3      | 0.1-0.3      |
| Selenium                    | (mg)    | 0.1-0.2     | 0.1-0.2      | 0.1-0.2      |
| Iodine                      | (mg)    | 0.1-0.3     | 0.1-0.3      | 0.1-0.3      |
| Vitamins <sup>2</sup>       |         |             |              |              |
| Vitamin A                   | (UI)    | 3450        | 3500         | 3500         |
| Vitamin D                   | (UI)    | 400         | 600          | 600          |
| Vitamin E                   | (UI)    | 7           | 10           | 10           |
| Thiamin B1                  | (mg)    | 1.7         | 1.5          | 1.5          |
| Riboflavin B2               | (mg)    | 2.8         | 4            | 4            |
| Niacin                      | (mg) pp | 8.5         | 12           | 12           |
| Panthotenic acid            | (mg)    | 3.3         | 4.8          | 4.8          |
| Pyridoxine B6               | (mg)    | 0.8         | 1.2          | 1.2          |
| Choline                     | (mg)    | 42          | 60           | 60           |
| Folic acid                  | (mg)    | 0.8         | 1.2          | 1.2          |
| Cyanocobalamine B12         | (µg)    | 8           | 12           | 12           |

<sup>1</sup>The recommended allowances for trace element are those presented by Meyer (1981) in Germany

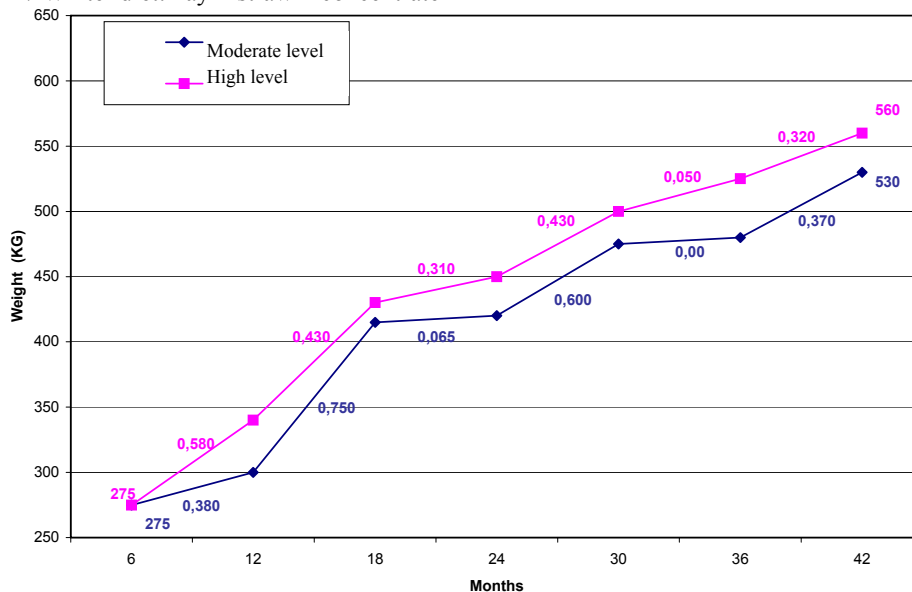
<sup>2</sup>The recommended allowances for vitamins are those presented by Wolter (1975)

Note: single concentrations values for some minerals, trace elements and vitamins are calculated for averages DM intake

## Conclusion

The targeted and allowed growth rate during the first winter (6-12 months) represents about 60 p100 of those achieved by young athletic horses devoted to competition and fed optimum nutrients requirements in order to exhibit their genetic potential (Figure 1). Energy and protein requirements represent respectively 80 and 75 p 100 of the optimum allowances (INRA, 1990).

A/ Winter diet: hay + straw + concentrate



B/ Winter diet: maize silage + concentrate

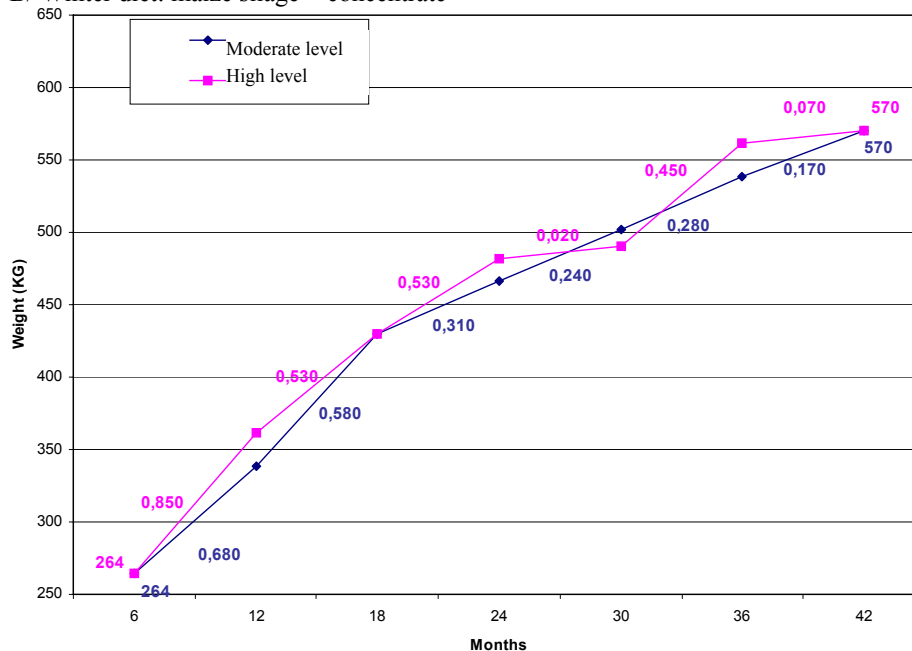


Figure 1. Evolution of the body weight from 6 to 42 months of age of growing saddle horses (AA-SF) fed dry forages (hay + straw) or maize silage based diets at two levels of intake (INRA – HN, 1979-1982; Bigot et al., 1987; Trillaud-Geyl et al., 1986).

During the second winter (18-24 months) targeted and allowed growth rate represents 40 to 50 p 100 of optimum growth rate achieved by athletic horses (Figure 1). Energy and protein allowances account for 85 and 80 p 100 of optimum allowances respectively (INRA, 1990).

During the third winter (30-36 months) growth rate represents only 30-40 p 100 of optimum growth rate achieved by athletic horses (Figure 1). Energy and protein allowances account for 90 and 80 p 100 of optimum allowances respectively (INRA, 1990).

Achieved winter growth rates, allow horses to reach between 40 to 48 months body weight and body size similar to those of athletic horses, as far as young horses are able to implement compensatory growth at pasture during summer season (Martin-Rosset *et al.*, 1984, Trillaud-Geyl *et al.*, 1990, 1992).

As a result horses devoted to leisure or even to be ridden for competing at moderate level, and breeding fillies can be managed using husbandry systems which maximise the intake of preserved forage during winter and green grass during summer periods (Figure 2).

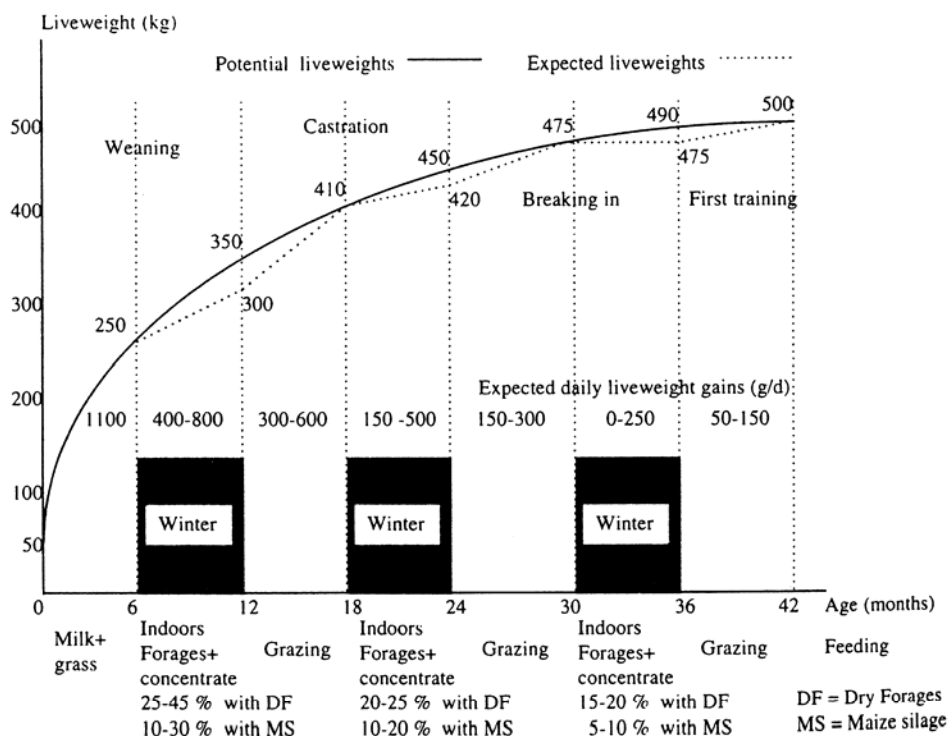


Figure 2. Feeding management of growing saddles horses (According to Bigot *et al.*, 1987; Trillaud-Geyl *et al.*, 1990; INRA, 1990).

These systems have been extensively validated in long term feeding experiments conducted both by the French National Studs and INRA (INRA, 1990).

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# Energy and protein requirements for growth: A European perspective

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## Introduction

### Energy

#### NRC

Energy requirements for growth are directly related to the energy content of the gain; it increases as the animal gets older, reflecting the changing proportions of tissues. Furthermore, rapid liveweight gain is usually associated with more energy being deposited per unit of gain reflecting increased fat deposition. NRC (1989) estimates the DE requirement per kg of gain as:

$$\text{Mcal DE kg}^{-1} \text{ LWG} = (4.81 + 1.17X - 0.023X^2) \text{ ADG} \quad (1)$$

[X is the age in months, and ADG is the average daily gain in kg]

This equation was developed from data reported by a number of different authors including Ott and Asquith (1986) and Schryver *et al.*, (1987). There is no optimum, defined rate of growth for horses or ponies although excessive weight gain has been associated with the development of bone abnormalities (Thompson *et al.*, 1988).

#### INRA

Two studies (Agabriel *et al.*, 1984; Bigot *et al.*, 1987) provided data on energy intake, liveweight and resulting liveweight gain in growing horses. This information was incorporated into a ruminant model:

$$\text{UFC kg}^{-1} \text{ W}^{0.75} \text{ day}^{-1} = a + bG^{1.4} \quad (2)$$

(Geay *et al.*, 1978; Robelin, 1979)

and used to predict energy requirements for growth. The exponent value 1.4 was validated by Agabriel *et al.*'s, (1984) work; 'a' represents the coefficient of maintenance requirement, 'b' the coefficient of gain and 'G' is the average daily gain (kg d<sup>-1</sup>). Table 1 gives the coefficients used for calculating UFC requirements.

Table 1. INRA (1990) coefficients for calculating UFC requirements for Growth.

| Age (months) | Coefficients |        |
|--------------|--------------|--------|
|              | a            | b      |
| 6-12         | 0.0602       | 0.0183 |
| 18-24        | 0.0594       | 0.0252 |
| 30-36        | 0.0594       | 0.0252 |

Table 2 shows a comparison between estimates made by NRC (1989) and Martin-Rosset *et al.* (1994) of the DE requirements of growing horses with a mature weight of 500 kg.

NRC (1989) appears to produce higher estimates of requirement although it should be noted that equation (1) is age-dependent rather than weight-dependent; the latter is likely to be a more reliable basis for calculating requirements.

Table 2. A comparison between INRA<sup>1</sup> (1990) and NRC (1989) estimates of the DE requirements of growing horses (mature weight 500 kg).

| Age (months) | Weight (kg) | Gain (kg d <sup>-1</sup> ) | System | MJ DE required d <sup>-1</sup> |        | Total MJ DE d <sup>-1</sup> |
|--------------|-------------|----------------------------|--------|--------------------------------|--------|-----------------------------|
|              |             |                            |        | Maintenance                    | Growth |                             |
| 6            | 215         | 0.85                       | NRC    | 32.80                          | 39.13  | 71.96                       |
|              |             |                            | INRA   | 32.89                          | 21.14  | 54.03                       |
| 12           | 325         | 0.65                       | NRC    | 46.65                          | 42.47  | 89.12                       |
|              |             |                            | INRA   | 44.84                          | 26.10  | 70.94                       |
| 18           | 400         | 0.35                       | NRC    | 56.07                          | 26.77  | 82.84                       |
|              |             |                            | INRA   | 52.39                          | 22.66  | 75.05                       |

<sup>1</sup> Assumes 1 UFC = 12.87 MJ DE

## Protein

### NRC

Deprivation of protein is probably no more damaging than deprivation of energy for horses as in other animals (Allden, 1970). Restricting protein intake will limit growth rate (Ellis and Lawrence, 1979) although foals born at the height of the grass-growing season in the UK will have available to them grass containing between 160 and 200g CP kg<sup>-1</sup> DM. Mare's milk that can provide up to 0.4 kg protein daily, together with good grass will support up to 1.5 kg gain d<sup>-1</sup> in thoroughbred foals; it is unlikely that these animals will be protein-limited. Based on an analysis of published work, NRC (1989) recommends 12 and 10.8 g CP MJ<sup>-1</sup> DE for weanlings and yearlings respectively. It is generally considered that lysine is the most limiting amino acid and that 502 and 454 mg should be supplied per MJ DE to weanlings and yearlings respectively.

### INRA

Probably the most reliable way to estimate protein need in growing animals is to conduct serial slaughter studies and subsequent tissue analysis. Martin-Rosset *et al.*, (1983) examined heavy breeds of horses during their growth phase and estimated protein requirements on the

basis of tissue composition. They assumed a faster turnover of body protein and increased the maintenance protein requirement for growing animals by 125% of maintenance i.e. 3.5 g MADC kg<sup>-1</sup> W<sup>0.75</sup>. Incidentally, Meyer (1983) allows 4.5g DCP kg<sup>-1</sup> W<sup>0.75</sup>. For light horse breeds, Martin-Rosset *et al.*, (1994) used a model similar to that used for determining energy requirements (Equation 3) to calculate total MADC requirements:

$$\text{g MADC day}^{-1} = a W^{.75} + bG \quad (3)$$

where 'a' is the coefficient of maintenance, 'b' the coefficient of gain and G is the average daily gain (kg d<sup>-1</sup>); these coefficients are shown in Table 3. Comparisons of estimated requirements for growth using the different systems (Table 4) shows that it is difficult to compare the values other than by making assumptions about the diets fed and the digestibility of the protein contained therein. The protein content of gain declines as liveweight increases; it is about 200g kg<sup>-1</sup> gain in young animals (3-6 months) and around 170g kg<sup>-1</sup> gain in a 2 year old animal with a mature weight of 500 kg.

*Table 3. INRA (1990) coefficients for calculating MADC requirements for growth.*

| Age (months) | Coefficients |     |
|--------------|--------------|-----|
|              | a            | b   |
| 6-12         | 3.5          | 450 |
| 18-24        | 2.8          | 270 |
| 30-36        | 2.8          | 270 |

*Table 4. Comparisons between INRA(1990) and NRC (1989) protein requirements of growing horses (mature weight 500 kg) together with estimated DCP requirements (gd<sup>1</sup>).*

| Age (months) | Weight (kg) | Gain (kg d <sup>-1</sup> ) | System | CP (g) | DCP <sup>1</sup> (g) | MADC (g) | DCP <sup>2</sup> (g) |
|--------------|-------------|----------------------------|--------|--------|----------------------|----------|----------------------|
| 6            | 215         | 0.85                       | NRC    | 860    | 602 <sup>3</sup>     | -        | -                    |
|              |             |                            | INRA   | -      | -                    | 579      | 579 <sup>5</sup>     |
| 12           | 325         | 0.65                       | NRC    | 958    | 575 <sup>4</sup>     | -        | -                    |
|              |             |                            | INRA   | -      | -                    | 560      | 609 <sup>6</sup>     |
| 18           | 400         | 0.35                       | NRC    | 891    | 535 <sup>4</sup>     | -        | -                    |
|              |             |                            | INRA   | -      | -                    | 345      | 375 <sup>6</sup>     |

<sup>1</sup> estimated from NRC data.

<sup>2</sup> estimated from INRA data.

<sup>3</sup> assumes 0.70 CP is digestible;

<sup>4</sup> assumes 0.60 CP is digestible;

<sup>5</sup> assumes concentrate only;

<sup>6</sup> assumes 50:50 concentrate/hay

## A comparison between European feeding systems and practises

### A theoretical comparison between systems for feeding youngstock

Martin-Rosset and Tisserand (2004) calculated rations based on good hay, barley and soya for a 12 month-old yearling, weighing 320kg and growing at 0.75kg d<sup>-1</sup>. Using NRC (1989) data (requirements and feed values), the animal would require 2.8, 3.54 and 0.66 kg (2.2%BW)

respectively of the hay, barley and soya per day whereas according to INRA (1990), the figures were 2.39, 3.12 and 0.39 kg (1.8% BW). These authors concluded that the TDMI was less, when the diet was based on INRA requirements and that this diet supplied between 16 and 24% less CP than the NRC-based diet for moderate (0.45 kg d<sup>-1</sup>) and rapid growth (0.75 kg d<sup>-1</sup>) respectively. The differences between the systems were still apparent when diets were constructed for 2 year-olds although the discrepancies were of a reduced magnitude. Cuddeford (1997) also concluded that NRC requirements for energy and protein were higher than those proposed by INRA. It appears that, when using the INRA system, less concentrate is required both in absolute amounts and in proportion to forage. One of the major criticisms levelled at the NRC system has been that it overvalues forages in terms of their putative contribution of energy and protein within a ration. However, in the light of the foregoing this would seem irrelevant as more food is fed in order to satisfy a higher requirement according to NRC guidelines. Examination of practical diets may indicate which system is closer to reality in the context of feeding the growing horse.

**A comparison between European feeding practices**

*UK*

A weanling (227kg), typically fed 1.8kg of a medium quality hay and 4.3kg of balanced compound feed at about six months of age would consume both energy and protein in excess to need using either feed system. A yearling (320kg) fed 4.0kg medium quality hay together with 3.2kg of yearling cubes, a long yearling (404kg) fed 4kg hay and 4kg compound or a two year-old (440kg) in training fed 4kg hay and 6kg racehorse cubes will all receive an excess of protein; irrespective of system, it appears that energy supply meets need fairly precisely. However, this is no real surprise as animals continuously respond to intakes of energy and thus, feeders will increase or decrease amounts of feed according to desired rates of gain or animal performance. It would seem that in the UK, growing Thoroughbreds are persistently overfed protein and, even replacing the roughage with poor quality hay, still leads to an oversupply according to NRC(1989) requirements (see Table 5).

*Table 5. Comparisons between energy and protein supply and requirements of different systems<sup>1</sup>.*

| Horse type    | Intakes |      |      | %NRC |     | %INRA |      |
|---------------|---------|------|------|------|-----|-------|------|
|               | DM%BW   | MJDE | gCP  | DE   | CP  | UFC   | MADC |
| Weanling(6m)  | 2.2     | 65   | 908  | 102  | 129 | 114   | 128  |
| Yearling(12m) | 2.0     | 70.4 | 872  | 105  | 152 | 100   | 110  |
| Yearling(18m) | 2.25    | 80   | 1000 | 115  | 185 | 100   | 140  |
| 2yearold      | 2.25    | 107  | 1080 | 105  | 119 | 100   | 165  |

<sup>1</sup> based on data presented by Harris (1999)

*Norway*

Studies with growing (0.6-0.8kg d<sup>-1</sup>), 8 to 9 month-old, Standardbred trotters (D. Austbø, personal communication) weighing 260/270 kg have produced some interesting comparative data as summarised in Table 6. It will be apparent from the table that the provision of a

‘straights-based’ diet-more “natural feeding”-results in a severe protein shortage to the growing animal.

*Table 6. Comparisons between different diets in terms of their ability to meet NRC (1989) requirements for energy and protein for growing Standardbreds.*

| Diet type                                  | Intakes |      | %NRC |     |
|--------------------------------------------|---------|------|------|-----|
|                                            | MJDE    | gCP  | DE   | CP  |
| 7kg hay/2kg concentrate                    | 86      | 938  | 109  | 96  |
| 9kg hay/2kg concentrate <sup>1</sup>       | 104     | 1108 | 131  | 112 |
| 7kg hay/0.5kg oats/1kg barley              | 81      | 756  | 103  | 71  |
| 9kg hay/0.5kg oats/1kg barley <sup>1</sup> | 99      | 926  | 125  | 86  |

<sup>1</sup> *Ad lib* roughage/outdoor exercise available 24h d<sup>-1</sup>

It is clear that these animals were consuming up to 4.2% of bodyweight; in excess of the intakes predicted by the NRC (1989) of 2.0 to 3/3.5% of bodyweight as air-dry feed. Less concentrate was fed to these growing Standardbreds than is normally fed to Thoroughbreds of the same age in the UK. As a consequence, there is less of an oversupply of protein; the higher intakes of feed energy by animals kept in groups outside is possibly a reflection of the sum of the effects of increased activity and a winter environment (very cold in winter ie., -20 °C).

### *Germany*

There appears to be a lack of information on the practical feeding of young Warmblood horses in Germany however, attempts have been made to calculate energy and protein intakes of foals by surveying the feeds available to these animals over a large number of farms (M. Coenen, personal communication) and using GEH (1994) requirements; these data are summarised in Table 7. It was found that there was a large degree of variation in the way in which foals are fed in the Hanover area. Contrasting these data with NRC (1989) requirements shows that in all cases, there is good agreement between energy supply and need. The foals are being underfed protein in the earlier stages of growth but this disparity reduces as the animals become older (see Table 8). This underfeeding of protein probably represents the fact that the roughages available to these animals are not of sufficient quality. This situation may be addressed by improving conservation techniques or switching to silages that are of inherently better quality (see Table 14). Alternatively, a less desirable approach would be to increase concentrate allowances or, the protein content of the concentrates available could be increased.

*Table 7. Calculated daily intakes of digestible energy (DE, MJ d<sup>-1</sup>) and digestible crude protein (DCP, g d<sup>-1</sup>) by Hanoverian foals from 2 to 6 month of age.*

| Age (months) | 2   | 3   | 4   | 5   | 6   |
|--------------|-----|-----|-----|-----|-----|
| Weight       | 120 | 155 | 187 | 215 | 240 |
| Weight gain  | 1.3 | 1.2 | 1.1 | 1.0 | 0.9 |

| (DE MJ d <sup>-1</sup> , DCP, g d <sup>-1</sup> ) | DE  | DCP | DE  | DCP | DE  | DCP | DE  | DCP | DE  | DCP |
|---------------------------------------------------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
| mean values                                       | 49  | 471 | 55  | 483 | 61  | 561 | 65  | 652 | 67  | 733 |
| n                                                 | 556 | 554 | 579 | 578 | 579 | 576 | 563 | 563 | 466 | 465 |

Table 8. Comparison of calculated intakes by growing Hanoverian foals with NRC (1989) recommendations<sup>1</sup>.

| Foal characteristics |        |                    | Calculated supply (Table 7) |                          | % NRC (1989)             |                          |
|----------------------|--------|--------------------|-----------------------------|--------------------------|--------------------------|--------------------------|
| Age                  | Weight | Kg d <sup>-1</sup> | DE (MJ d <sup>-1</sup> )    | DCP (g d <sup>-1</sup> ) | DE (MJ d <sup>-1</sup> ) | DCP (g d <sup>-1</sup> ) |
| 4                    | 187    | 1.1                | 61                          | 561                      | 94                       | 80                       |
| 5                    | 215    | 1.0                | 65                          | 652                      | 96                       | 96                       |
| 6                    | 240    | 0.9                | 67                          | 733                      | 97                       | 113                      |

<sup>1</sup> assumes mature weight of 650kg and that crude protein is 70% digestible

### Netherlands

Dutch Warmblood foals may be reared intensively or extensively, the former usually for sale at a foal auction at 4 months of age. Table 9 shows that the supply of energy and protein relative to requirement is excessive in most cases relative to CVB (1996) requirements and also NRC (1989). Thus, we may conclude that within the Dutch system of rearing Warmbloods there is a real danger of overfeeding, excessive weight gain and possibly an increased disposition to DOD.

Table 9. A comparison between feed supply and requirements (CVB, 1996) for energy and protein by Dutch Warmblood foals at different ages, growing at different rates to reach a mature size of 500kg.

| Age (m)         |     | BW(kg) Rations (kg fresh weight) |              |              |              |                           | %CVB(1996) |      |
|-----------------|-----|----------------------------------|--------------|--------------|--------------|---------------------------|------------|------|
|                 |     | Grass                            | Grass silage | Maize silage | Foal pellets | Milk (l d <sup>-1</sup> ) | VEP        | VREp |
| 3 <sup>1</sup>  | 160 | 9                                | 2            | 0            | 1            | 17                        | 148        | 120  |
| 6 <sup>2</sup>  | 235 | 0                                | 8            | 2            | 2            | 0                         | 97         | 78   |
| 12 <sup>3</sup> | 335 | 45                               | 0            | 0            | 0            | 0                         | 163        | 114  |

<sup>1</sup> intensive rearing for foal auction

<sup>2</sup> extensive rearing, group housed

<sup>3</sup> spring grass with no supplementation

### Italy

It is apparent from Table 10 that, in most cases, energy requirements are met which is to be expected as animals will regulate growth rate in response to energy inputs. However, protein supplies are deficient in the rations of young, growing horses relative to NRC (1989) estimates of need. A comparison with INRA (1990) requirements for MADC for either a 6 month weanling or a 12 month yearling thoroughbred indicates a supply of about 100% of requirement in each case.

### France

Hay-based diets have been the traditional way of feeding growing horses in France although, in common with many other European countries, silage-based diets are becoming more popular. In particular, diets are increasingly being based on multi-wrap, big or small bale silages and are characterised by being of high dry matter content, usually in excess of 500g/kg

compared to conventional big bale silages for cattle of about 300g kg<sup>-1</sup>. Table 11 shows a range of diets fed to Anglo-Arabs at a national stud (Trillaud-Geyl, personal communication) where groups receive both types of diet and where silage has been fed since 1992. Silage diets, as expected, supply more protein than hay-based diets that are inadequate in this respect (see Table 12).

*Table 10. A comparison between the supply of energy and protein and their requirement (NRC, 1989) by growing Thoroughbreds and Sella Italiano horses in Italy.*

| Animals and diets   |        |        |                                  | Energy and protein supplied d <sup>-1</sup> |      |       |      |
|---------------------|--------|--------|----------------------------------|---------------------------------------------|------|-------|------|
| Type                | BW(kg) | Age    | Diets(kg)                        | DE(MJ)                                      | %NRC | CP(g) | %NRC |
| T <sup>1</sup> bred | 220    | w'ling | 4H <sup>1</sup> /3C <sup>2</sup> | 66                                          | 105  | 680   | 90   |
|                     | 330    | y'ling | 5H/4.5C                          | 91                                          | 102  | 940   | 98   |
|                     | 470    | 2yo    | 6H/4.5C                          | 98                                          | 102  | 1020  | 100  |
| Sella Italiano      |        |        |                                  |                                             |      |       |      |
|                     | 300    | w'ling | 5H/3.5C                          | 79                                          | 98   | 820   | 74   |
|                     | 450    | y'ling | 6.5H/5C                          | 108                                         | 100  | 1120  | 87   |
|                     | 590    | 2yo    | 7H/5C                            | 112                                         | 100  | 1160  | 103  |

<sup>1</sup> first cut hay

<sup>2</sup> concentrates

*Table 11. A comparison between hay- and silage-based diets fed to different ages of Anglo-Arab horses.*

| Anglo-Arab characteristics |            |                          | Diets (kg d <sup>-1</sup> ) |                     |       |             |
|----------------------------|------------|--------------------------|-----------------------------|---------------------|-------|-------------|
| Age(m)                     | Weight(kg) | LWG(kg d <sup>-1</sup> ) | Hay                         | Silage <sup>1</sup> | Straw | Concentrate |
| 6-12                       | 270-330    | 0.6                      | 6.0                         |                     |       | 2.0         |
| 18-24                      | 430-460    | 0.25                     | 5.5                         |                     | 3.0   | 3.0         |
| 30-36                      | 500-510    | 0.05                     | 7.5                         |                     | 2.5   | 3.5         |
| 6-12                       | 280-350    | 0.6                      |                             | 9                   |       | 3.5         |
| 18-24                      | 440-485    | 0.38                     |                             | 16.5                |       | 4.0         |
| 30-36                      | 530-550    | 0.25                     |                             | 15.5                |       | 3.5         |

<sup>1</sup> DM-450g kg<sup>-1</sup>

*Table 12. A comparison between hay- and silage-based diets in terms of their ability to supply energy and protein to Anglo-Arab horses relative to their requirement (NRC, 1989).*

| Animals and diets |         |                          | Energy and protein supplied d <sup>-1</sup> |      |       |      |
|-------------------|---------|--------------------------|---------------------------------------------|------|-------|------|
| Diet              | Age (m) | LWG(kg d <sup>-1</sup> ) | DE(MJ)                                      | %NRC | CP(g) | %NRC |
| Hay-based         | 6-12    | 0.6                      | 67                                          | 100  | 660   | 83   |
|                   | 18-24   | 0.25                     | 91                                          | 100  | 865   | 95   |
|                   | 30-36   | 0.05                     | 109                                         | 100  | 1045  | 95   |
| Silage-based      | 6-12    | 0.6                      | 83                                          | 100  | 912   | 92   |
|                   | 18-24   | 0.38                     | 117                                         | 100  | 1380  | 118  |
|                   | 30-36   | 0.25                     | 112                                         | 101  | 1260  | 112  |

## Impact of forage quality on meeting energy and protein needs

Norwegian roughages are largely grass-based and hay is the most popular form of conserved grass although high DM silages (mean value 530g kg<sup>-1</sup>) are becoming increasingly popular (D. Austbø, personal communication). He has summarised the data for 2002 as shown in Table 13. It is clear from the table that proportionally, more silages are of better quality than are hays; this is to be expected as their making is not so dependent on a continuum of good weather.

Table 13. Characteristics of Norwegian hays and silages made during 2002.

| Hays<br>(% of n)<br>n=309 | Silages<br>(% of n)<br>n=126 | DE<br>(MJ kg <sup>-1</sup> DM) | UFC<br>(kg <sup>-1</sup> DM) | CP<br>(kg <sup>-1</sup> DM) | DCP<br>(kg <sup>-1</sup> DM) |
|---------------------------|------------------------------|--------------------------------|------------------------------|-----------------------------|------------------------------|
| 15                        | 43                           | >10.5                          | >0.66                        | 163                         | 119                          |
| 29                        | 35                           | 9.9-10.5                       | 0.61-0.66                    | 141                         | 98                           |
| 36                        | 16                           | 8.9-9.8                        | 0.55-0.60                    | 110                         | 68                           |
| 19                        | 6                            | 7.3-8.8                        | 0.45-0.54                    | 99                          | 58                           |
| <1                        | 0                            | <7.3                           | <0.45                        | <99                         | <58                          |

A survey of roughages produced for horses in Germany (M. Coenen, personal communication) compared grass silages with hays (Table 14). The mean pH of 99 silage samples was 5.8 with a minimum of 4.6 and a maximum of 7.6 with a DM in excess of 600g kg<sup>-1</sup>. The mean crude protein of these silages was 124g kg<sup>-1</sup> DM compared to that of hays that had a comparable value of 100 although, more than half of the sample values fell well below this.

Table 14. Proportion of hays and silages<sup>1</sup> containing different levels of crude protein from the Hanover region of Germany.

| gCP kg <sup>-1</sup> DM | % of Silages | % of Hays |
|-------------------------|--------------|-----------|
| <60                     | 3.9          | 7.4       |
| 60-80                   | 15.8         | 24.1      |
| 80-100                  | 14.5         | 25.9      |
| 100-120                 | 23.0         | 16.7      |
| 120-140                 | 19.7         | 13.0      |
| 140-160                 | 14.5         | 9.3       |
| 160-180                 | 5.3          | 1.9       |
| >180                    | 3.3          | 1.9       |

<sup>1</sup> n = 98 samples in each category

A soon to be published paper (Bergero *et al.*, In press) provides information on the chemical characteristics and apparent digestibility of some Mediterranean roughages. This Italian work showed that natural meadow hays contained between 647 and 698 g NDF kg<sup>-1</sup> DM, 70 to 85 g CP kg<sup>-1</sup> DM (only 33 to 44g DCP kg<sup>-1</sup> DM) and had an apparent OM digestibility of between 536 and 424g kg<sup>-1</sup> DM. These high fibre hays, together with their low content of available protein, are unsuited to feeding to young, rapidly growing animals; significant protein supplementation will be required.



Samples of horse hays and horse ‘haylages’ (high dry matter, wrapped bales of silage) sent to Dodson & Horrell Ltd (the major UK horse feed manufacturer) by horse owners for analysis are representative of material fed to horses throughout the UK. Generally, their analytical profiles follow those of roughages analysed in other Northern European countries, demonstrating the superior quality of silages over hays in terms of both protein and energy content. This reflects the fact the grass for haylage production is cut at a younger stage of growth than grass destined to be made into hay; in the UK, the climate is notoriously fickle and long spells of settled, fine weather are not the norm thus, production of quality hay is not favoured. UK grass hays rarely contain more than 80g crude protein kg<sup>-1</sup> and most would be around 60 with a depressing number of samples below 50. Haylage samples that were analysed contained, on average, around 120g crude protein kg<sup>-1</sup> DM, with lows of 100 and highs of 160, the precise figures obviously depending on the maturity of the material ensiled.

## Discussion

It is apparent from the foregoing that the practice of feeding growing animals varies throughout the EC and that it depends largely on the type of horse that is being reared. Thoroughbreds are fed little roughage and large amounts of concentrate whereas Standardbreds are more reliant on roughage. It seems that the desired rate of growth governs the relative proportions of roughage and concentrate; the higher the expected growth rate, the more concentrate is fed. In this context it is interesting to note that keeping horses on good, young spring grass will support high growth rates (see section 2.2.4). A common feature is that roughages (mostly hay) are an inadequate source of protein for growing foals. The fact that not enough crude protein is supplied is one factor but perhaps more importantly, it is doubtful if the quality of the protein will be good enough to support adequate growth. Feeding excess energy relative to protein has been shown to result in poorer body condition in growing foals (Ellis and Lawrence, 1979); energy restriction prior to grass growth/availability can lead to compensatory growth and possibly increased risk of DOD. The weight of evidence suggests that provision of more protein of moderate quality than that originally proposed as necessary by NRC (1978) results in increased growth rates. In contrast, INRA (1990) proposes an MADC value of ~66% of NRC (1989) for a 6 month weanling and ~54% of NRC (1989) at 12 months of age. If we can assume MADC to be ~70% of crude protein then clearly the INRA (1990) system suggests that less protein is required by the growing animal. As yet we are not in a position to objectively assess which system is the better in this regard. NRC (1989) suggests that weanlings require 50g CP Mcal<sup>-1</sup> DE d<sup>-1</sup>, yearlings require 45g CP Mcal<sup>-1</sup> DE d<sup>-1</sup> and 2 year-olds need 42.5g CP Mcal<sup>-1</sup> DE d<sup>-1</sup>. INRA (1990) recommends 118g MADC UFC<sup>-1</sup> d<sup>-1</sup> at 6 months, 107g at 12 months, 62g for 2 year-olds; this represents a much greater decline in the protein: energy ratio than with NRC; converted to MADC Mcal<sup>-1</sup> DE these figures become 37g, 34g and 20g MADC Mcal<sup>-1</sup> DE. Converting back to crude protein using a factor of 0.70 the comparable figures are 53g, 49g and 29g CP Mcal<sup>-1</sup> DE d<sup>-1</sup> however, these data are suspect in view of the approximations and the assumptions that are made to convert between systems. The ratios that exist in practical diets are shown in Table 15 and whilst the UK follows the trend shown by NRC (1989), French and Italian data suggest that protein is being underfed relative to energy in diets for growing horses in these countries.

Table 15. A comparison of the protein: energy ratios (g CP MJ<sup>-1</sup> DE) used in diets for growing horses in the EC relative to NRC (1989) values.

| Age (m) | NRC<br>(1989) | Country |                          |       |                 |                    |         |
|---------|---------------|---------|--------------------------|-------|-----------------|--------------------|---------|
|         |               | UK      | Netherlands <sup>1</sup> | Italy | France<br>(hay) | France<br>(silage) | Germany |
| 6       | 12.0          | 14.0    | 16.7                     | 10.3  | 9.8             | 11                 | 15.6    |
| 12      | 10.8          | 12.4    | 9.0                      | 10.3  | ?               | ?                  | ?       |
| 24      | 10.2          | 10.0    | ?                        | 10.4  | 9.5             | 11.8               | ?       |

<sup>1</sup> assumes 76VEP/MJ DE and that CP is 70% digestible

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## **Part C**

### **Minerals and vitamins requirements**



# The physiological role of minerals and vitamins in the growing horse

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## Introduction

The body composition of the new-born foal, young and the adult horse (Schryver *et al.*, 1974; Meyer and Ahlswede, 1976; Webb and Weaver, 1979; Grace *et al.*, 1999) reflects the accretion of macro and trace elements in different compartments of the equine body. However, the measurable amounts of minerals give a rather fragmentary figure about the physiological role and the growth related metabolism. It should be mentioned, that a simple quantitative figure (e.g. vitamin E in muscle) is a rather weak information concerning the importance for the process of growth.

A second point - and even more relevant if we look on reproduction - is that the impact of mineral and vitamin supply to the growing organism is not initiated when we observe or measure the growth after birth, in fact we have to consider some importance of these nutrients in the organism for successful fertilisation and the following stages of intrauterine life. Therefore the idea of this paper is to draw attention on the several stages of growth and the impact of some nutrients on several processes. The presented facts are partly not obtained in experiments in the equine species. But data from other species are well suitable to describe a certain function. Therefore the authors refer to the contribution of Kienzle (see page 199) presenting the quantitative figures about requirements and recommendations.

## Intrauterine and postnatal growth

The fertilised egg cell needs a specific environment for cell division and the intrauterine ambience is characterised among others by minerals and vitamins. As summarised in Figure 1 macro and trace elements are essential constituents of the intrauterine environment for establishing the pregnancy.

In that context it should be recognised that the fertilisation rate in horses underlying a well organised breeding programme is ~95 % (Morris and Allen, 2002), and the main losses in pregnancy occur up to day 35. That reflects the sensitivity of the early stage of pregnancy. Of course, the processes after fertilisation are complex. A loss of pregnancy during the first ~ 30 days do not simply indicate a deficit in one of the listed nutrients (Figure 1). But the ionic composition of the fluid in the oviduct as well as in the uterus differs from that in plasma (at least in pig and cattle, Seiwald, 2001). In conclusion, there must be a specific system to establish that condition. Changes in intrauterine pH and ammonia concentration after different protein intake in cows shows the possible influence of nutrition on the conditions for the ovum (Wiebold *et al.*, 1988; Elrod and Butler, 1993; Elrod *et al.*, 1993; Butler *et al.*, 2002). Furthermore in vitro-studies show the influence of minerals and vitamins on the development of the ovum towards the blastocysts (Seiwald, 2001). Although the 2-cell or later phases of embryonic life is a pretty model to study the influences of nutrients up to now the present results are not sufficient to describe precisely the essentiality of macro-, trace elements and vitamins for early embryonic life particular in horses.

| phase               | element         | vitamin          |
|---------------------|-----------------|------------------|
| before implantation | Ca              | Vit. A, E, B12   |
|                     | Fe, Cu, Zn, Co, | cholin           |
|                     | J, Mn, Se       | folic acid ,     |
|                     | Cd, Pb, Ni      | pantothenic acid |
| after implantation  |                 | riboflavin       |
|                     | Ca              | inositol         |
|                     | Cu, Zn, Se, J   | Vit. A, E, B12   |
|                     | Co, Fe          | folic acid       |
|                     | Cd              | pantothenic acid |
|                     |                 | riboflavin       |
|                     |                 | inositol         |

Figure 1. Factors influencing the development of fertilized egg cell in several mammalian species (in modification from Ashworth and Antipatis, 2001; Hostetler et al., 2003).

After implantation the embryo becomes more and more dependent on the nutrient exchange crossing the placenta. There seems to be a certain dimension of the placenta surface in relation to the foetus (Figure 2) to optimize the nutrient transfer from maternal to foetal side. The nutrient transfer from the mare to the foetus peaks during the late stage of gestation, when the main weight gain occurs (Figure 3). The foetus in principle seems to be rather safe during the later pregnancy. The survival of lambs from ewes highly affected by pregnancy ketosis demonstrates that the maternal organism compensates the insufficient intake by mobilisation of reserves independent of disadvantages for the ewe.

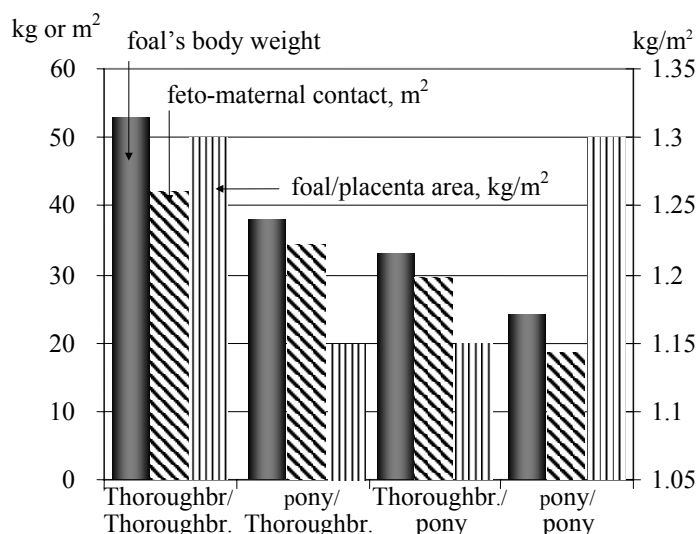


Figure 2. Intra-uterine conditions and foetal development – effects of mating in Thoroughbreds and ponies for different combinations (Allen et al., 2002a).



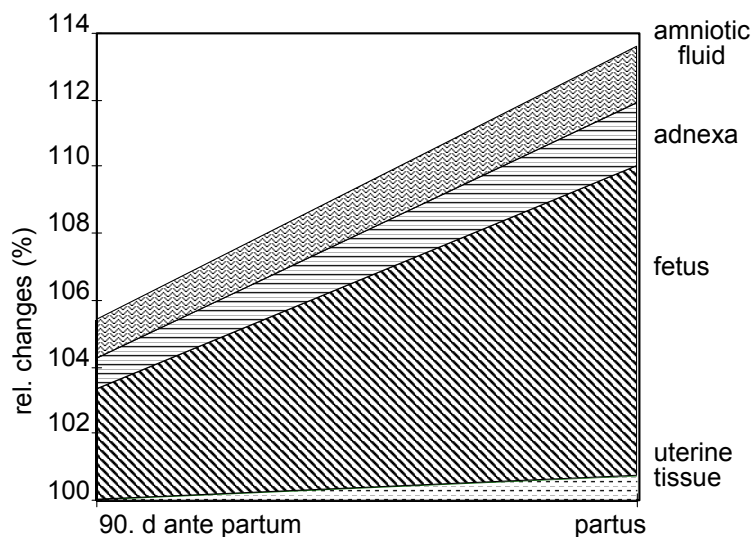


Figure 3. Weight changes in the mare during the last 3 month of gestation (in relation to body weight after birth).

But the reality is that a shortage in nutrients is well recognized by the maternal and foetal organism. Feeding ewes a low calcium diet from d 60 of gestation resulted in delayed foetal skeletal ossification although the plasma components of calcium homeostasis in the foetal lambs were not affected (Lima *et al.*, 1993). A low calcium intake of pregnant mares is found to reduce intrauterine weight gain of the foals, probably by depressed bone mineralization (Austbø and Dolvik, 1996). It can be assumed that not all effects which are generated throughout gestation are compensated after birth. A vitamin A deficiency is still world wide one of the most important nutrient deficiencies in human and combined with a deficit in iron. A low vitamin A status during pregnancy affects foetal growth and the health status of the newborn (Basu *et al.*, 2003, Gazala *et al.*, 2003). Reduced organ weight, changed tissue properties (lowered elastic fibres), delayed lung maturation and reduced gene expression are effects of a low maternal vitamin A intake (Antipatis *et al.*, 2000). These effects are established in the foetus and cannot be compensated after birth. The vitamin A status (including  $\beta$ -carotene) in horses is rather safe under natural conditions with fresh pasture. But in cases with low  $\beta$ -carotene intake (stabled horses, no supplements and hay of poor quality) exists the risk for a lowered reproduction rate and a supplementation is recommended (Schubert *et al.*, 1991).

For the foal copper is a prominent example for the micronutrient intake of the mare and the consequences in the foetus (Meyer, 1994). Copper is stored in the foetal liver in relation to mare's copper intake (Figure 4). After birth the foals need these copper stores for the initial growth as the milk is low in copper. A low copper intake in the pregnant mare therefore will reduce the copper status of the newborn foal.

These examples demonstrate that the intrauterine part of growth is clearly influenced by nutritional factors particular by the intake of micronutrients. Negative effects may persist throughout the postnatal growth and influence the health of the young horse just as it occurs in other mammalian species.

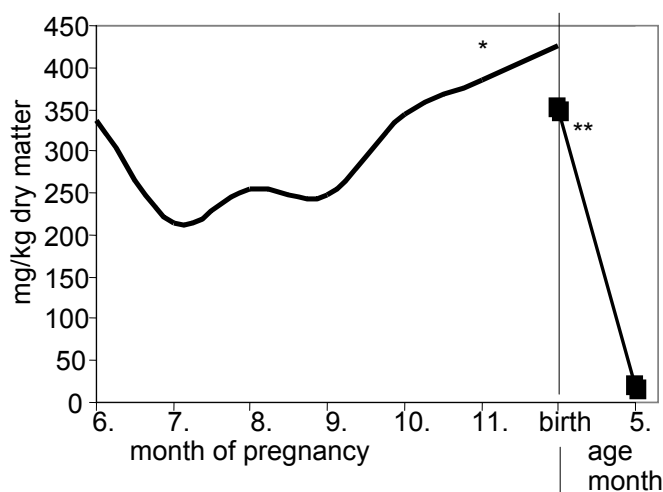


Figure 4. Copper in the equine foetal liver and in foals after birth (Hebeler et al., 1996\*; Van Weeren et al., 2003\*\*).

## The hormonal framework

Already in the early stages of the blastocyst there is a role for growth related endocrine activities. The myometrial activity of the uterus is influenced by parathyroid hormone (PTH) and the processes resulting in implantation are influenced by PTH related protein. The genes regulating the production of PTHrP are located in the uterine epithelium (Wysolmersky *et al.*, 2002). From those facts can be concluded that growth includes the maturation of the endocrine system (Allen *et al.*, 2002b) of the entire organism and that the endocrine control over growth is part of mineral metabolism. Figure 5 gives the principles of the endocrine network.

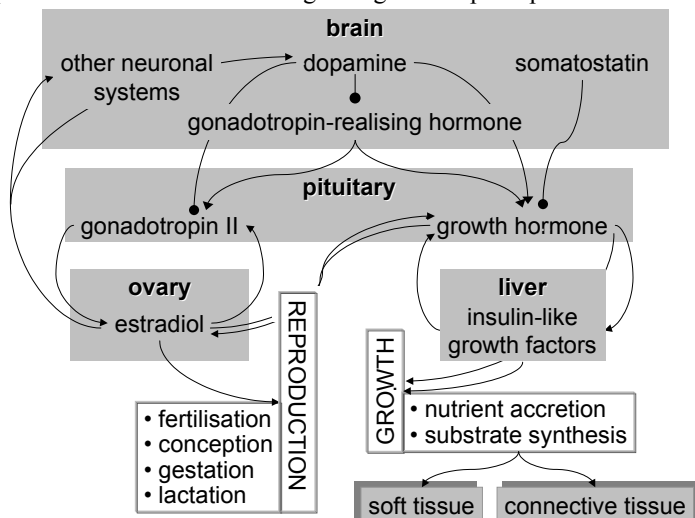


Figure 5. Integrated control of growth related endocrine factors (accordingly Blázquez *et al.*, 1998, modified after Wysolmersky *et al.*, 2002 and Karaplis, 2002).

stimulation → inhibition ◆

The minerals participate in that concert not only directed to growth and accretion of certain minerals in tissues which realize specific gain, but also regarding processes in the background of growth (Table 1).

*Table 1. Selected factors and selected effects on mineral metabolism.*

|                              | calcium         | phosphorus        | magnesium         | sodium            |
|------------------------------|-----------------|-------------------|-------------------|-------------------|
| main depot                   | skeleton        | skeleton          | skeleton          | skeleton          |
| GH, IGF-1                    | in tissues ↑    | in tissues ↑      |                   |                   |
| TGF- $\alpha$ , TGF- $\beta$ | in tissues ↑    | in tissues ↑      |                   |                   |
| TH                           | in bone ↑       | in bone ↑         |                   |                   |
| EGF                          |                 | renal excretion ↓ | tissue uptake ↑   |                   |
| PTH, PTHrP                   | in bone ↑ and ↓ | in bone ↑ and ↓   | renal excretion ↓ | renal excretion ↑ |
| calcitonin                   | in blood ↓      | blood P ↓         |                   |                   |
| insulin                      | in bone ↓       | in bone ↓         | tissue uptake ↑   |                   |
| 1,25(OH) $_2$ D              | absorption ↑    | absorption ↑      | absorption ↑      |                   |
| gastrin                      | in blood ↓      |                   |                   |                   |

Abbreviation: PTH = parathyroid hormone, PTHrP = parathyroid hormone related peptide, TGF= transforming growth factor, GH = growth hormone, IGF-1 = insulin like growth factor-1, TH = thyroid hormone, EGF= epidermal growth factor

Growth associated hormones like GH or IGF-1 act in regulation of processes with synthesis of tissue mass including bone. In consequence, if a mineral (e.g. Ca) is deposited in bone, there is a link to GH which is involved in bone growth. Not all aspects are sufficiently investigated, e.g. the main part of potassium is located in muscle tissue. It can be assumed that there is an influence of GH on potassium import into the growing muscle. Of course that needs to be confirmed in specific studies. Beside of growth determining and regulating hormones there are others which influence mineral balance. One of the most exciting hormones is PTH which has an effect on the osteoblasts in two different directions, a) the catabolic effect by stimulation the osteoclasts via activated osteoblasts and b) the anabolic side by the enforced activity of osteoblasts without transmission to the osteoclasts (Hodsman *et al.*, 2002).

Regarding the intensive feeding of young horses and the start of training at the premature status (e.g. in Thoroughbreds) the negative effect of insulin on bone Ca- and P-balance is of great interest. The clinical site of this interaction is investigated in some detail by Ralston (1996). After glucose load healthy foals showed a clearly lower glycemic and insulinemic response in comparison to those foals which had osteochondrotic lesions (Figure 6).

Although outside of the hormone family 1,25-dihydroxy-cholecalciferol [1,25(OH) $_2$ D] is introduced in Table 1 as an important factor for mineral balance and as a vitamin integrated in the hormone based regulation of growth. For the equine species there are some uncertainties about the role of vitamin D. The sensitivity against high doses responsible for calcinosis is comparable to other species, but the sensitivity against a low intake resulting in rickets obviously is lower e.g. to swine or dog.

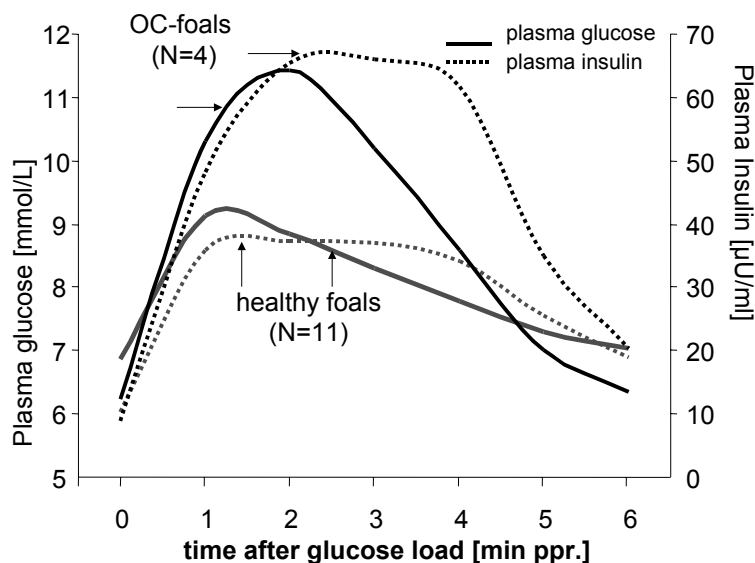


Figure 6. Plasma glucose and insulin after glucose load in healthy foals and foals with osteochondrotic lesions (Ralston, 1996).

## Macro elements

### Calcium and phosphorous

First digestion trials concerning calcium and phosphorous were done 1836 by Boussingault (Jean-Baptiste Joseph Dieudonné Boussingault, 1801-1887, professor for chemistry in Lyon and later for agricultural science in Paris). But detailed investigations in mineral metabolism were still rare. In contrast to that numerous reports already from the 19<sup>th</sup> century draw attention on a widely distributed disease in horses (although in part fed naturally on grass which today often is argued to be a sufficient feed for horses covering any nutrient requirement) known as “big head disease” and later described as adult rickets (for review see Ohlendorf, 1998). Not until the beginning 20<sup>th</sup> century feeding trials were conducted to reproduce the disease by changing Ca- and P-intake. The bulk of research regarding these minerals was done after 1960 and is actually focussed on aspects of calcium transport and ion channels as well as on the bone health complex.

The main portion of body calcium is deposited in bone (99.8 % of total, Grace *et al.*, 1999). Outside of the skeleton calcium is present in membranes and circulating substrates. In addition to the contribution to ossification, the element is involved in blood clotting, muscle contraction, enzyme activities related to energy metabolism and digestive processes and transmission of neural signals (Wijnberg *et al.*, 2002). Calcium is absorbed in the small intestine at about 50–80 %, obviously by a passive pathway (Schryver *et al.*, 1970, Meyer *et al.*, 1982, Telep, 1984). The secretion in the hindgut results in an overall absorption of about 45 %, in general nearly 20 % (absolute) below the prececal net absorption. In contrast to ruminants the absorption rate from the intestine is rather independent from Ca-intake and a surplus of absorbed Ca is eliminated via urine (Figure 7).

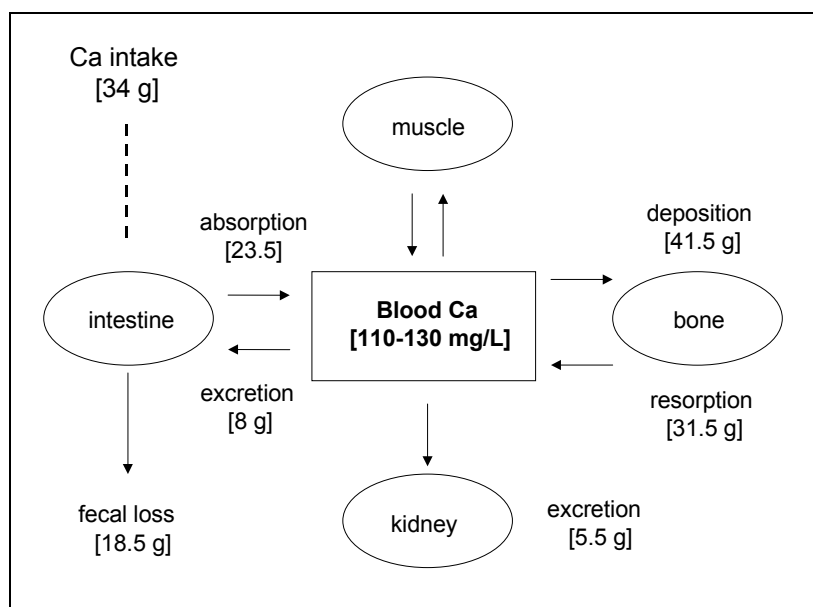


Figure 7. Calcium metabolism in adult horses (500 kg BW, daily Ca intake of 34 g).

Consequently, the renal Ca-excretion alone or the creatinine:calcium-ratio in urine can be used to evaluate the Ca-intake (Table 2, Meyer and Stadermann, 1990).

Table 2. Renal output of minerals and creatinine:mineral ratio in horses (Meyer and Stadermann, 1990).

| mineral |                      | dimension                | quality of intake |         |          |
|---------|----------------------|--------------------------|-------------------|---------|----------|
|         |                      |                          | marginal          | low     | adequate |
| Ca      | absolute             | mg/kg BWxd <sup>-1</sup> | <4                | 4-8     | >8       |
|         | Cre <sup>1</sup> /Ca | mg/mg                    | >7.5              | 7.5-3.5 | <3.5     |
| Mg      | absolute             | mg/kg BWxd <sup>-1</sup> | <4                | 4-8     | >8       |
|         | Cre <sup>1</sup> /Mg | mg/mg                    | >7.5              | 7.5-3.5 | <3.5     |
| Na      | absolute             | mg/kg BWxd <sup>-1</sup> | <3                | 3-6     | >6       |
|         | Cre <sup>1</sup> /Na | mg/mg                    | >10               | 10-5    | <5       |
| K       | absolute             | mg/kg BWxd <sup>-1</sup> | <10               | 10-20   | >20      |
|         | Cre <sup>1</sup> /K  | mg/mg                    | >3                | 3-1:5   | <1:5     |
| Cl      | absolute             | mg/kg BWxd <sup>-1</sup> | <5                | 5-10    | >10      |
|         | Cre <sup>1</sup> /Cl | mg/mg                    | >6                | 6-3     | <3       |

<sup>1</sup> creatinine and element in urine

Vitamin D seems to be less important for modifying absorption than in ruminants (Breidenbach *et al.*, 1998). On the other side like other species the horse shows changes in bone development in response to vitamin D depletion (El Shorafa *et al.*, 1979). Vitamin D-metabolism in the horse still needs more precise figures to derive safe data for formulation of requirements.

The Ca-homeostasis includes the protein-bound fraction, the ionized Ca and at a small degree complexed calcium. The concentration of  $\text{Ca}^{++}$  in blood is the main trigger for the hormone system. A drop in plasma  $\text{Ca}^{++}$  during high intensive exercise is answered by an increase in PTH (Vervuert *et al.*, 2002), but this response is not sufficient to ensure a total upregulation of  $\text{Ca}^{++}$  in blood by mobilisation from bone (Figure 8).

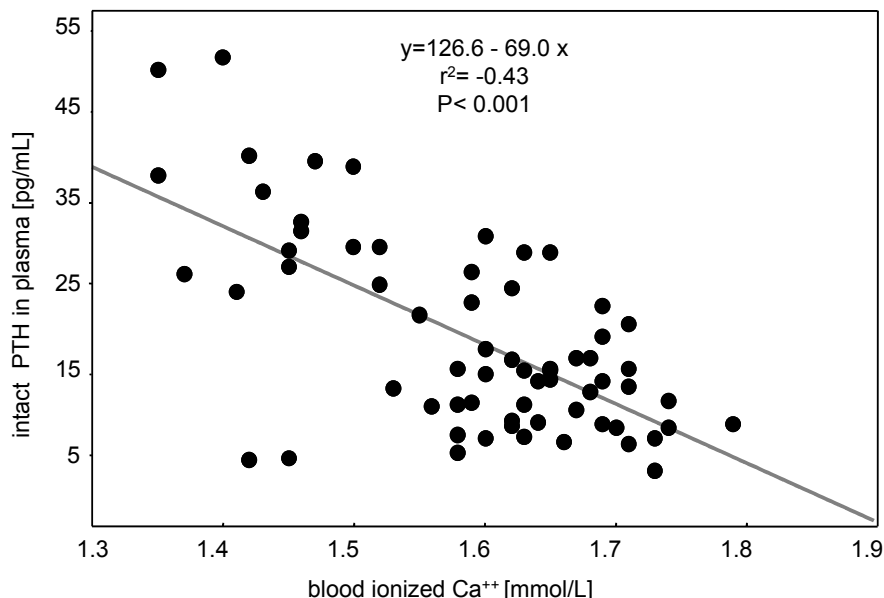


Figure 8. Correlation between blood ionized  $\text{Ca}^{++}$  and intact PTH during exercise (Vervuert *et al.*, 2002).

The counterpart of PTH is represented by calcitonin, which is activated at high calcium intake. In several species calcitonin depresses gut absorption, mobilisation from bone and reabsorption by the kidneys. The role of calcitonin in the horse is not sufficiently investigated. From the facts that absorption in the equine intestine is not sensitive against calcium intake and that 1,25-dihydroxycholecalciferol ( $1,25\text{-(OH)}_2\text{D}$ ) is low in the horse, it can be concluded that calcitonin in the equine gut is less effective than in other species and that the interaction between hormones like PTH and calcitonin, and  $1,25\text{-(OH)}_2\text{D}$  is less delicate. But again, that conclusion must be confirmed. The activity of the calcium-binding-protein (strongly involved in calcium absorption) is remarkably depressed by glucocorticoids and the effect is an age dependent decrease in calcium absorption (Glade, 1982). Therefore the independence of calcium absorption from calcium intake do not implicate the insensitivity of absorption against any interacting principle.

A high calcium intake induces a rise in gastrin secretion as shown in rat, ruminant, dog, poultry, and human (Luthman *et al.*, 1975, 1977; Floor *et al.*, 1991; Persson *et al.*, 1991; Coenen *et al.*, 1997; Rümenapf *et al.*, 1997) by the elevated blood gastrin concentration. An increase in gastrin on the other site is accompanied by a hypocalcaemia (Rümenapf *et al.*, 1998). The calcium-reducing principle obviously is not gastrin itself, but is located in the oxyntic gland area of the stomach. A gastrectomy or prolonged depression of the gastric acid secretion can induce a loss in bone density and bone mineral content. These results are obtained from e.g. rats, humans and poultry and seem to represent a general principle

(Rümenapf *et al.*, 2000). In summary, these interactions show that a high calcium intake is not only a question of entire balance. Particular in the growing horse the “risk assessment” of calcium overdosage needs to take into account the impact on the hormone system.

The bulk of phosphorus in bone (80.8 % of total, Grace *et al.*, 1999) deposition is similar to calcium, and in addition organic phosphorus is indispensable for numerous processes beside bone and therefore present in nearly all tissues. The contribution of phosphorus to energy metabolism as a constituent of ATP and in forming RNA and DNA reflects the universal role of that element.

Phosphorus is a constituent of several secretions in the small intestine. That results prececally in a net secretion in dependence on the roughage (TELEP, 1984) intake which increases the secretion (e.g. saliva). In the hindgut occurs a secretion as well as absorption. The different localised sites of calcium and phosphorus absorption, small intestine and hindgut respectively, explain the limited role of the calcium:phosphorus ratio in the horse. The focus of absorption of phosphorus in the hindgut is logical as we understand the equine species as a member of the hindgut-fermenter-family. Phosphorus is essential for microbial digestion. A low phosphorus availability depresses ruminal microbial N-synthesis and digestibility of organic matter (Durand *et al.*, 1983; Komisarczuk *et al.*, 1987; Breves, 1991). Komisarczuk *et al.* (1987) conclude that a range of 75-100 mg phosphorus/l rumen fluid maintain the maximum degradative and synthetic microbial activities. The phosphorus concentration in the ileal chyme of the horse varies at ~0.65 g/kg (Meyer *et al.*, 1982) and will provide sufficient amounts of that element to the microbial community. In addition, the high phosphorus concentrations in secretion (horse saliva ~ 20 mg/l, Coenen, 1986, equine bile juice 200 mg/l, Meyer, 1992) will ensure phosphorus flux from the small intestine to the cecum. Matsui *et al.* (1999, phosphorus intake 21-22 g/d) measured a daily total phosphorus passage in Thoroughbreds at ~ 40-60 g in the lower part of the small intestine and ~83 in the upper large intestine. A further benefit of the absorption in the hindgut is the independency of non-phytate-phosphorus in the horse. Phytate phosphorus is sufficiently available for the horse due to the microbial breakdown of that phosphorus source (Matsui *et al.*, 1999). The big variation in absorption in dependence on phosphorous intake, source and type of ration results in an unpredictable renal excretion. In contrast to other macro elements there is no safe inference on intake by renal phosphorous excretion or the creatinine:phosphorus ratio.

There is a comparable regulation network for phosphorus as for calcium particular considering the role of both elements for bone formation (see beneath). In principle vitamin D elevates phosphorus absorption, but as described above concerning calcium, the role of vitamin D in the horse is not well investigated. Under marginal phosphorus intake in small ruminants the transport mechanisms for phosphorus modify their efficiency to compensate in part the deficit or surplus of the element (Huber, 2003). By hypothesis one could project that onto the horse. That is beside the contribution of phosphorus to bone growth an exciting context. The intestinal secretion coming up after birth would be associated with a phosphorus depletion, which would be deleterious for an animal with an immediate demand to realize successful flight, if there is no capacity (already in the newborn) to save phosphorus. A comparable situation exist for the goat in contrast to pig. For goats it is described that the ontogenesis forming the transport capacities particular in the kidneys to save phosphorus by reabsorption is finished throughout the intrauterine life.

Both elements represent the majority of anorganic substance in bone. They are organized in an unique crystalline structure with high mechanical resistance but being still a living tissue,

that means the persistency of bone “construction” and bone degradation belong to the biological properties of bone. For the equine species, which survived by reason of her capacity to flight immediately after birth it is essential that bone formation is going on throughout the intrauterine life parallel to the ontogenesis and the processes which are involved in bone growth must already mature in the foetus. That is confirmed by result obtained in foals. The bone markers in blood show a decrease during the first week of life, whereas PTH is independent on age (Vervuert *et al.*, 2004).

Bone is formed by collagen fibres (type I, 90 % of total protein) and non-collagenous proteins. Spindle or plate shaped crystals of hydroxyapatite ( $\text{Ca}_{10}[\text{PO}_4]_6[\text{OH}]_2$ ) are found on the collagen fibres and in the ground substance. The mechanisms of mineralization are not fully understood. The ground substance is composed of glycoproteins and proteoglycans and these highly anionic complexes have a high ion-binding capacity and are thought to play an important role in the calcification process and the fixation of hydroxyapatite crystals to the collagen fibres. Osteocalcin, a non-collagenous protein which is synthesised by the osteoblasts is thought to affect the growth or maturation of  $\text{Ca}^{++}$ -phosphate mineral phases. However, Mg plays also an important role in mineralization as Mg has been shown to bind to the surface of hydroxyapatite crystals and seems to compete with calcium for the same absorption site (Rude, 2002). In addition, high Mg content in the diet resulted in smaller crystals and affect the perfectness of the crystal structure.

## Magnesium

At the beginning of the 20<sup>th</sup> century magnesium was found to be essential for animals. The knowledge about the clinical site of magnesium deficiency including hypomagnesaemia and tetany in horses is merely ~70-75 years old (Montgomery *et al.*, 1929; Sjollem, 1930; Kruse, 1932; Green, 1935).

The net absorption of magnesium over the entire GIT-tract varies between 5 and 60 % (Hintz and Schryver, 1972; Mundt, 1978). The main absorption occurs in the small intestine (~ 60 %), which is about double the total absorption (Meyer *et al.*, 1982). In conclusion, under physiological condition a magnesium secretion occurs in the hindgut. But in principle magnesium can be absorbed in the large intestine if the concentration in the chyme is elevated (e.g. by rectal infusion). A linear relationship between magnesium intake ( $x$ , mg/kg BW $\times$ d<sup>-1</sup>) and faecal excretion ( $y$ , mg/kg BW $\times$ d<sup>-1</sup>) =  $0.51 + 0.54x$ , von Bieberstein, 1990) indicates the independency of absorption from intake. Consequently the surplus of ingested magnesium is excreted via urine. The regression equation  $y = 2.18 + 0.17x$  ( $n=107$ ,  $r=0.72$ , Meyer and Stadermann, 1990) describes the relation between magnesium intake ( $x$ , mg/kg BW $\times$ d<sup>-1</sup>) and renal output ( $y$ , mg/kg BW $\times$ d<sup>-1</sup>).

About 60 % of magnesium is stored in the skeleton, 32 % in muscle tissue (Grace *et al.*, 1999). In bone magnesium is associated to the crystalline structure (see above) and the incorporation uses the same regulation as calcium and phosphorus. Most of the magnesium outside of bone is located in the intracellular space. Here it is associated to specific structures like mitochondria or substrates like ATP, RNA or DNA. Most metabolic processes (e.g. oxidative phosphorylation, protein synthesis) inside the cells need magnesium, which is one of the most abundant intracellular cations. The dysfunction of the magnesium dependent ATPase attached to myosin is part of the mechanisms which create the clinical appearance of magnesium deficit.



Magnesium deficiency is a widely distributed problem in pregnant women particular in rural areas (Pathak *et al.*, 2003). In veterinary medicine hypomagnesaemia in cows is the most prominent complication in relation to low magnesium intake. The classic form occurs on pasture with high concentration in soluble nitrogen and potassium. These factors interfere with ruminal magnesium absorption. The indoor magnesium deficiency becomes more important. In comparison to the ruminants magnesium metabolism in the equine is less stressed by low intake. The magnesium concentrations in roughage average at ~1.5 g/kg dry matter. In the growing horse, intrauterine as well as postnatal magnesium deficiency (producing clinical signs) is obviously a rare condition in contrast to exercising horses. But the view on the main routes in the discussion about magnesium neglects the important interaction of that element with the immune system. That is not sufficiently investigated in the equine and therefore the impact of magnesium on horse's health status is difficult to evaluate. Independent of species, a magnesium depletion is associated with an increased level of proinflammatory cytokines (Tam *et al.*, 2003). Increased lipid peroxidation and plasma nitric oxide in hearts of magnesium deficient rats (Bussire *et al.*, 2002) keeps magnesium in contact to the oxidative stress defence mechanisms. In addition, a dislocation of magnesium into the extracellular space seems to be as deleterious as the deficiency. Interestingly magnesium induced in *in vitro* studies with placenta cultures placental degeneration by a stimulation of apoptosis (Black *et al.*, 2001). In that context it should be mentioned that in uterine flashings from dairy cows with abnormal embryos magnesium concentrations are higher in comparison to samples from cows with normal embryos. The association of intrauterine growth retardation with elevated magnesium concentrations in amniotic fluids is a further clue for the need of a specific distribution of magnesium inside and outside of cells (Facchinetti *et al.*, 1989). However, it needs some research in horses to elucidate the evidence of these interaction.

### **Sodium, potassium and chloride**

The mammalian body is a watery system in which all reactions are organised. The watery environment is characterised by an specific acid-base-balance and osmotic condition. The electrolytes sodium, potassium and chloride are not exclusively responsible in that concern, but indispensable. The benefits of feeding salt to horses are known since ~210 years (Wolstein 1794, see Ohlendorf, 1998 for review). Nutritional aspects of potassium were first described at the end of the 19<sup>th</sup> century. The main experimental work in horses regarding these macro elements is done during the last 40 years, mainly focussed on the exercising horse by reason of the high losses in these elements via sweat.

The skeleton contains ~ 51 % of body sodium while potassium is allocated to muscle by 75 % (Lindner, 1983; Gürer, 1985). Chloride is distributed over several tissues in the range of 10-15 % of total body chloride (Coenen, 1991). For sodium and chloride exist a high secretion in the prececal part of the GIT. Both are effectively absorbed in the hindgut and net absorption over the entire GIT is ~95 % (Meyer *et al.*, 1982; Coenen, 1991). In contrast to chloride, potassium is absorbed in the small intestine (~52-74 %, Meyer *et al.*, 1982), and a secretion follows in the hindgut and total tract net absorption varies <50 %. The faecal output of sodium and chloride is independent of intake while faecal potassium excretion increases at a limited degree in relation to intake. For all electrolytes exists a linear relationship between intake and renal excretion. The modification of renal output is all dominant adaptation to high or low intake. The control system differs totally from that for the other macro elements and is summarised in Figure 9, which presents the hypothalamus-pituitary-adrenal-axis.

As described above, the ovum prior and after fertilisation needs a conducive condition in the uterus. The osmolarity in the fluids of the oviduct and uterus depends on these electrolytes, which are quite different concentrated in comparison to plasma (Seiwald, 2001) and show also remarkable differences between species. Expanding the fluid compartment in the growing foetus as well as after birth includes the increase in the amounts of electrolytes but there is no dominating structure like for calcium and phosphorus build up by the electrolytes. The regulation of water and electrolytes (Figure 9) ensures a proper hydration status and ionic composition of the tissues. The hypothalamo-pituitary-adrenal-axis is active already in the foetus (Butler, 2002) and stress sensitive.

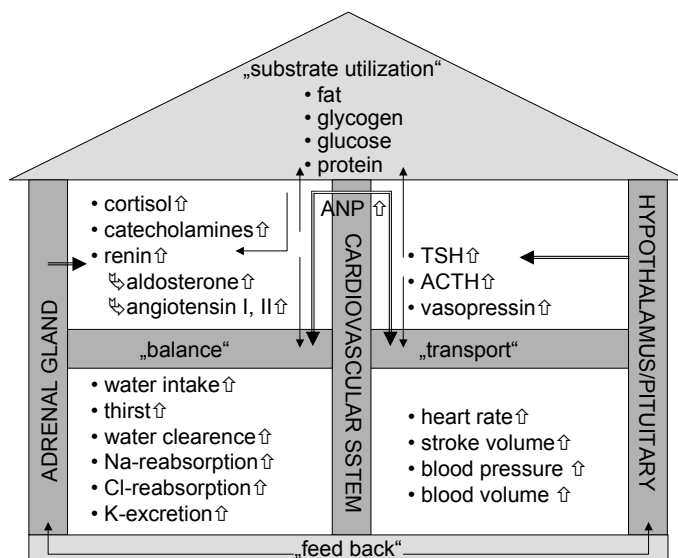


Figure 9. Regulation of selected reactions associated with exercise in the equine by the "hypothalamic-pituitary-adrenergic-axis" schematic description.

⇒ secretion, → influence e.g. on water intake

ACTH=adrenocorticotrophic hormone; TSH=thyroid stimulating hormone, ANP =atrial natriuretic peptide (Coenen and Vervuert, 2003).

Not at least the electrolytes are essential contributors for several transport systems (e.g. Na-K-pump, glucose transport). Without these transport systems the equilibrium between different compartments and the energy metabolism would collapse. Chloride is part of different channels, one of the most exciting topics in channel research. Dysfunction of some chloride channels give reason for fatal bone disease like osteopetrosis. Malfunctions occur obviously mainly in relation to genetic aberrations but less by reason of insufficient supply.

There is increasing challenge for renal function in maintaining water and electrolyte balance in late gestation. The increase in blood volume, effective renal blood flow and glomerular filtration rate on maternal side is embedded in hormonal control including the atrial natriuretic peptide as major component (Dunlop, 1981; Olsson *et al.*, 1989; Irons *et al.*, 1996). That concept will protect the foetus against changes in maternal haemodynamics and electrolyte metabolism.

A rather direct influence of electrolytes on growth is the alkalizing or acidifying effect of a cation or anion excess, respectively. Regardless that the impact on acid base balance is not the property of a single electrolyte, but of the balance of cations and anions, it needs to be discussed here (for reviews see Eur. J Nutr. 2001, Vol. 40, issue 5). Sodium, potassium and chloride are not the only ions acting on acid base balance, but they represent the most important strong ions in that context. In addition, the variation in potassium concentration of feeds creates big changes in cation-anion balance and chloride dominates the anion side as it is one of the most important anions. The consequences of changing the ratio of cations:anions on systemic pH is well described and established in dietetic measures to prevent milk fever in dairy cows (increase anion intake = mild metabolic acidosis), to depress the frequency of the metritis-mastitis-agalactia-syndrome (anions  $\uparrow$ ) or to handle urolithiasis in ruminants, dogs and cats (anions  $\uparrow$  or bicarbonate to elevate urine pH in dependence on type of calculus). The increase in blood ionized calcium in cows after feeding additional anions (ammonium-chloride) shows the influence of the cation:anion-balance on calcium metabolism (Coenen *et al.*, 1997). In the background is a reaction of bone. An alkalosis depresses calcium mobilisation from bone and stimulates the collagen synthesis (Figure 10). In contrast to that an acidosis creates a calcium drainage from bone into the circulation (Figure 11). The foetus should be protected against the common influences on maternal acid-base-balance. A foetal acidosis in consequence of an rise in blood lactate during strenuous exercise or after surgical manipulation shows that the systemic acid-base-balance of the foetus can react (Taylor *et al.*, 1992; Nordstrom *et al.*, 2001) even in the foal. But it is not sufficiently investigated if the mare's reaction on changes in cation:anion-balance is an imprint for the foetus. However, from the principle regulation of acid-base-balance in different species it can be concluded that after birth there is the same response in the foal as in adults. Changing from milk diet to a roughage based diet throughout the first month of life will change the cation:anion-balance for the young horse. We can expect that under most conditions an alkalizing stimulus will be present by reason of the increased potassium intake. That could simulate the osteoblasts and depress the activity of osteoclasts. Detailed research is requested to elucidate these interactions with special regard to a low calcium intake under alkalotic circumstances.

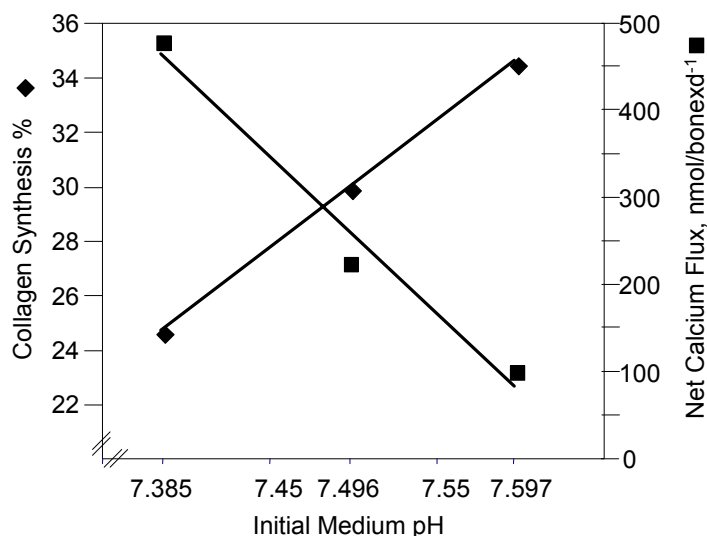


Figure 10. Calcium fluxes from mouse calvariae and collagen synthesis ( $[^3\text{H}]$ proline incorporation in % of total  $[^3\text{H}]$ proline) in dependence on pH in the incubation medium (Bushinsky *et al.*, 1996).

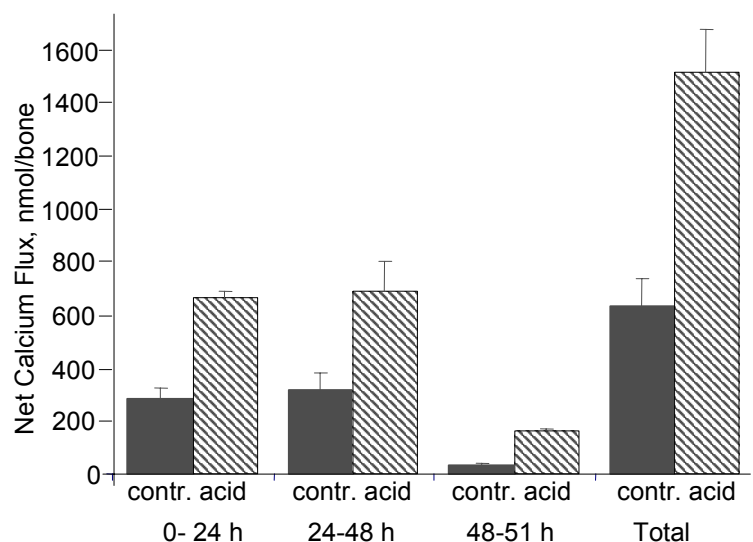


Figure 11. Net calcium fluxes from mouse calvariae incubated at pH ~7.45 (control) or 7.14 (acid) for totally 51 h (Bushinsky et al., 2003).

## Trace elements

The essentiality of iron and iodine was already discovered in the 18<sup>th</sup> and 19<sup>th</sup> century (Ohlendorf, 1998). Surprisingly that took place for the other trace elements rather late during the last two trimester of the 20<sup>th</sup> century. In some cases experiences in the toxicity are much older than the insight in the role as an essential element for life (e.g. selenium). Essential functions of the trace elements are listed up in Table 3.

Table 3. Trace elements and their main biological function.

| element | absorption side           | organotropism                                                             | function                                                              |
|---------|---------------------------|---------------------------------------------------------------------------|-----------------------------------------------------------------------|
| Cu      | small intestine           | liver, blood, spleen, lungs, brain, bones                                 | component of metalloenzymes (e.g. lysyl oxidase)                      |
| Zn      | small intestine           | pancreas, liver (metallothionein), pituitary gland, kidney, adrenal gland | activator of several enzymes for division, growth and repair of cells |
| Fe      | small intestine           | erythrocytes, liver, kidney, egg yolk complexed to heme                   | oxygen transport, heme compounds, heme enzymes                        |
| Mn      | small intestine           | bone, liver, kidney pancreas                                              | enzyme activator, constituent of metalloenzymes                       |
| Se      | small and large intestine | kidney, liver, spleen, pancreas                                           | Se-dependent glutathione peroxidases, several selenoproteins          |
| I       | GIT, lungs, skin          | thyroid, ovaries, pituitary gland, salivary gland                         | synthesis of thyroid hormones (thyroxine, triiodothyronine)           |
| Co      | small intestine           | muscle, bone, kidney, liver                                               | component of vitamin B <sub>12</sub>                                  |

## Copper

Copper is mainly absorbed in the small intestine at ~ 30-50 % (Meyer *et al.*, 1982), the total tract absorption varies between 40 and 50 %. The absorption may be higher in the young than in the adult horse. Key for absorption and sensitive for copper intake is the metallothionein in the intestinal epithelial cells. The absorbed element is mainly protein bound transported to the liver, the central organ for copper metabolism, in which synthesis occur. Ceruloplasmin is one of the most important products as it represents the carrier of copper to different tissues. Furthermore, copper is introduced in several enzymes.

Copper functions are involved in cellular respiration as it is a constituent of the cytochrome oxidase and in haemoglobin synthesis (McDowell, 1992; Anke *et al.*, 1994; Underwood and Suttle, 1999).

Copper is needed for bone formation and collagen synthesis. In this tissues the copper dependent enzyme, the lysyl-oxidase, is responsible for the formation of crosslinks, which gives the mechanical property of proteins in connective tissues (Meyer, 1994). A comparable function is fulfilled in the keratinisation process.

The cytochrome oxidase determines the hepatic phospholipid synthesis and the myelin formation. By that axis copper interferes with the nervous tissues. Dysfunctions therefore are visible by ataxia. Degenerative processes in the central nervous system seem to include copper, but the precise mechanisms are not clear up to now. In equine motor neuron disease a higher copper concentration was found in the spinal cord in comparison to healthy horses (Polack *et al.*, 2000). Copper is involved in the defence of free radicals by super oxide dismutase, but the accumulation obviously is not associated to an increased capacity to degrade free radicals. The same enzyme contributes to phagocytosis. By that way the element is linked to the immune system.

The wide spread functions of copper let expect an impact on function associated with growth. Reproductive failures in cases of copper deficiency confirm that link (Meyer, 1994, 1998). The role of copper like that of other trace elements is already mentioned in Table 3. During the later gestation foetal cell respiration and growth of bone and connective tissue is in the foreground (Meyer, 1994). A specific role of copper supply to the foetus is shown in copper storage in the liver (Meyer, 1994, see Figure 4). Although there is no doubt that a copper deficiency can produce orthopaedic problems in the horse, it's role in the aetiology of osteochondrosis is not valid (Coenen *et al.*, 2003; Van Weeren, 2003).

## Zinc

Zinc absorption is focused in the equine small intestine (~ 30-45 %), whereas a secretion is coming up in the hindgut (Meyer *et al.*, 1982). The absorption over the entire GT varies in dependence on ration over the range of <10 up to 25 %.

Zinc uptake from the GIT-tract is regulated by metallothionein which is sensitive to the presence of zinc. Protein bound zinc is delivered in the liver and released to extra hepatic tissues. There is a certain role of zinc for secretion processes. Therefore the pancreas and liver are the organs with the highest zinc concentrations.

Zinc is a nearly universal constituent of numerous enzymes in tissues of high metabolic activity like liver and protein synthesising tissues. The proper function of nucleic acids depends on zinc and by reason of that zinc influences the cell metabolism at all. Functions of zinc concern tissue growth but also synthesis of controlling substances like hormones and immunoglobulins. The benefit of zinc supplementation in children suffering by diarrhoea underline the role of zinc for immune status and health (Roy *et al.*, 1999; Dardenne, 2002) which should be taken into account for feeding ill animals.

Some evidence is given for the link between mental capacity in the offspring and maternal zinc intake in humans. Low zinc consumption by the mother during the pregnancy depresses learning abilities which might be related to a teratogenic effect in nervous tissue during embryogenesis. It is unknown if there is a comparable defect in the foal. Possibly detrimental effects on behaviour (drinking of the foal) can be part of the consequences (The effect of deficits in brain development are visible in mice by swim tests. That shows the impact of defects in central nervous tissue on the organism at all). Of course the most important consequence of a deficit in zinc is the depressed function of the DNA and RNA synthesis. In addition to these general interaction between zinc and growth there are some specific links of that trace element to growth processes in reproduction. Obviously, zinc is needed for the conceptus before maternal recognition of the pregnancy. In porcine conceptus zinc is higher concentrated than in the surrounding tissues (Hostetler *et al.*, 2000). That requires a special zinc drainage into the embryo in these early stages. The arachidonic cascade producing prostaglandins (e.g. PGF<sub>2α</sub>) is zinc sensitive (Sakuma *et al.*, 1999). A lack in PGF<sub>2α</sub> inhibits the establishment of pregnancy, at least in pig (Kraeling *et al.*, 1985). In that way zinc possibly interacts with the embryonic survival (see above pregnancy losses during the first 35 day in horses).

Later on, zinc can influence the binding properties of the insulin-like growth-factor (IGF-I and II) to receptors. This is essential for cell differentiation during any kind of growth coming up with cell cycles. Zinc shortage depresses the availability of IGF-I, what is a growth depressing condition (MacDonald, 2000).

In consequence, phases with high growth rate are accompanied with a high need in zinc. The mare has to cover the zinc demand for the intensive intrauterine growth and in addition for the accumulation of zinc in the liver. Here the zinc concentrations increase from <200 in 6<sup>th</sup> month of pregnancy up to >300 mg/kg dry matter at the end of gestation (Hebeler *et al.*, 1996).

## Iron

Although iron is not rare in humans and animals habitat, iron deficiency is a common disease in animals. In humans iron deficiency reflects – beside vitamin A deficit – the most important problem in nutrient supply and is connected to a lack in well being particular in children (Merialdi *et al.*, 2003). That is a confusing fact as the empirically based knowledge in iron as a nutrient is more than 2000 years old.

A few data only are present for iron absorption the horse. The prececal absorption is about 42-71 % (Teleb, 1984). The absorption is influenced by source of iron and iron status of the animal. The absorption of iron can be described like a spring controlled valve: a high iron status in the organism keeps it closer, a low iron status elevates the flow drastically. The dependence of iron absorption from intake is shown by classic experiments in rats

(Fairweather-Tait and Wright, 1984, Figure 12) and can be taken as a principle for many species. That is a logical principle for an element which is highly variable in supply and requirement and the tolerance for a shortage, even for a short termed deficit, is low. The renewal rate of erythrocytes and synthesis of iron containing enzymes need continuous available iron.

Iron ( $\text{Fe}^{++}$ ) is converted inside the mucosa cell to ferritin and oxidised after entering the blood stream to the ferric form, which complexes with transferrin. In that formation iron is distributed in the several tissues.

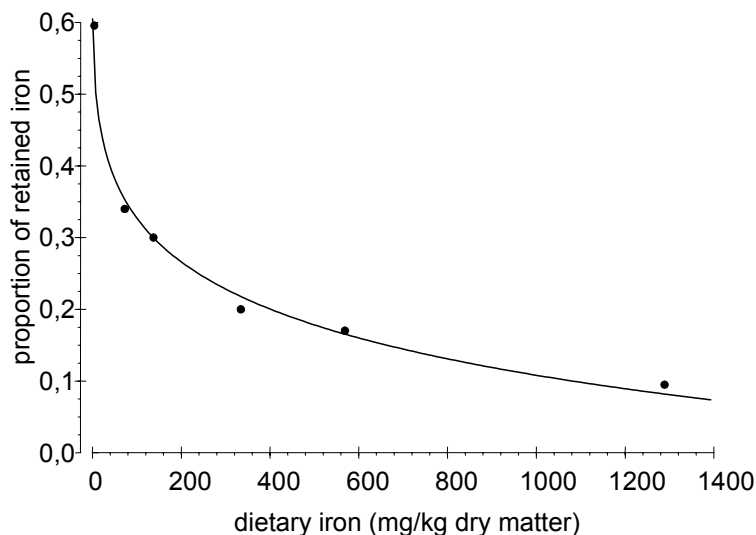


Figure 12. Total body iron retention in dependence on dietary iron in rats (Fairweather-Tait and Wright, 1984).

The main directions are bone marrow for haemoglobin synthesis, cells containing iron dependent enzymes and – in case of pregnancy – the placenta for transferring iron to the foetus. Iron covers functions in the cytochrome system (production of ATP), activation of oxygen and oxygen transport via haemoglobin (presenting ~ 60 % of total iron) or myoglobin. Therefore, biological active iron is protein bound, mainly as hemoprotein. The synthesis of hemoproteins is a complex procedure requiring copper. Iron containing enzymes are widely distributed in all types of tissues and in part excreted by milk.

Iron deficiency is directly linked to anaemia, loss in cell respiration and failure of the tricarboxylic acid.

The iron accretion in the foetus follows the development of foetal mass throughout the last month of pregnancy but there is no iron storage strategy like in case of copper. As the newborn foal requires a sufficient oxygen delivery to the muscle and enzyme profile, the mare needs to supply the pregnant uterus with increasing amount of iron and to ensure a sufficient iron concentration in milk.

Finally, iron depletion of the foetus by low maternal iron intake obviously lowers the cognitive capacity in humans (Pollit, 1993). Long lasting experiments with horses are

necessary to investigate this with regard to behaviour and training responses (see above for comment concerning brain development).

## **Manganese**

Absorption of manganese is comparably distributed over the GIT-tract as for copper, an absorption in the small intestine (~ 20-45 %), a secretion in the hindgut, and finally a total tract absorption of <10 up to ~50 % (Meyer *et al.*, 1982).

After transfer across the mucosal cells manganese is bound to proteins and transported to the liver. From here manganese is distributed throughout the body, the majority bound to transferrin. In the cells manganese is incorporated in the mitochondria. Neither high nor low intake of manganese induces big changes in tissue manganese concentrations. The manganese in liver, kidney, pancreas, muscle and bone is less mobile in comparison to other elements. Difference between new-born and adult animals are uncertain. Lambs and calves respond with a sharp increase in liver manganese in contrast to adult animals (Howes and Dyers, 1971, Watson *et al.*, 1973).

Manganese contributes to proper cell function by activation of enzymes or availability of manganese dependent enzymes and possibly plays a role for maternal pregnancy recognition by interacting with the secretion of progesterone in the first days after fertilisation (Hostetler *et al.*, 2003). One of three isoenzymes of superoxide dismutase (SOD) contains manganese. This enzyme is responsible for cell protection against damage by free radicals. This capacity is not only requested after birth, e.g. in case of muscle activity but also during the early embryonic and later foetal life. Bovine embryos present the Mn-SOD (Lequarre *et al.*, 2001), which is also effective and important for lung development in the foetus. Manganese has a unique function for bone growth, and bone is the most responsive tissue for dietary manganese. The enzymes for synthesis of mucopolysaccharides of bone require essentially manganese (Leach, 1967). As the synthesis of the organic matrix of bone is initiated before mineralization processes become relevant, there may be an increased demand for manganese already in mid pregnancy. But the transfer rate from the maternal to foetal side seems to be sufficient. During pregnancy there is no increase in liver manganese of the foetus in cows (Abdelrahman and Kincaid, 1993, GRAHAM *et al.* 1994) and the accumulation of 0.3 mg/d in Holstein Friesian cows balances the requirement which is low in comparison to intake.

## **Selenium**

Selenium is absorbed in the small intestine and in the hindgut (Wolfram, 2000). The absorption in monogastric animals is quite high (~ 70 %). As stated for the other trace elements, after absorption the transport of selenium occurs bound to a protein. In contrast to the trace elements mentioned up to now there is no homeostatic control of absorption which makes a high selenium supply as dangerous. One could conclude that under natural conditions which defined the selection of the equine species no mechanism was necessary to protect the organism against high selenium (see iron or copper) and that the equine is well adapted to low selenium supply.

Rich in selenium under physiological conditions are liver and kidneys. The contribution of selenium to the glutathion-peroxydase (GSH-Px) is well known and mostly discussed as the primary function of that element. As muscle dystrophy is a selenium responsive disease, selenium is usually described in association with the integrity of muscle. The GSH-Px is an



important factor in cell protection against free radicals. In the defence of cell membranes against peroxides that enzyme co-operates with vitamin E. As peroxides generating lipid oxidation occurs in early stages of embryonic life and of course later on, when the foetus increases in body mass, selenium is of importance for reproduction (Segersen *et al.*, 1977). However, the essentiality of selenium in that direction for the horse is not sufficiently investigated. Icelandic Horses in Iceland, a native, endemic breed, are well adapted to a low selenium intake. These horses have low blood selenium concentrations and low GSH-Px-activities, but they do not develop diseases, which are related to a low selenium intake like muscle disorders or reproductive failure (Vervuert *et al.*, 2001). On the other hand, myopathy can be produced by deficient selenium and vitamin E supply in guinea pigs (Hill *et al.*, 2001).

The role of selenium in the immune response seem to be of more importance. Selenium protects macrophages during phagocytosis and improves the immunological reaction. That is mirrored by the inverse relationship between selenium intake and the incidence of mastitis in dairy cows (Hogan *et al.*, 1990). The improvement of the immunocompetence by elevated selenium intake is confirmed also for horses (Knight and Tyznik, 1990). Baalsrud and Overnes (1986) observed an elevated antibody production in Thoroughbreds after feeding additional selenium.

A selenium-dependent enzyme - known for ~ 14 years only - that catalyses the deiodination of L-thyroxine to the biological active hormone is of great interest with regard to growth. That function could be in the foreground for the growing horse even throughout the foetal period, whereas the risk of muscle diseases like white muscle disease by reason of a simple selenium deficit seems to be overestimated. Selenium easily crosses the placenta and is available for foetal growth. As the thyroid function is involved in the determination of gestation and contributes to the signal for birth, selenium interacts with this processes at the end of pregnancy. The role for the immune system is relevant after birth when the contact with the new environment asks for sufficient defence mechanisms. As the foal (obviously similar to the calf, Abdelrahman and Kincaid, 1993) is born without mobile selenium reserves to be available for that task, it needs selenium supply by the milk. The concentration of that trace element in milk is determined by selenium intake of the dam (Anke *et al.*, 1994; Wolfram, 2000).

## **Iodine**

Iodine is as a constituent of the thyroid hormones, thyroxine and triiodothyronine. However, the importance of these hormones for growth is in disagreement with the iodine research in horses. Iodine crosses different epithelia (skin, lung, GIT). A high absorption therefore can be expected. There exists a selective iodine uptake of the absorbed iodine by the thyroid tissue. The iodine in thyroid tissue and kidney including urine covers nearly the total body iodine pool. The thyroid gland stores the iodine compounds and the iodinated hormones. They are involved in energy exchange in all cells, interact with other hormone producing tissues and maturation of many tissues and the neuronal control. So far, these hormones represent a omnipotent principle and are the key for growth.

The placenta ensures the iodine and thyroid hormone supply to the foetus by an active transport system. The thyroid hormones are active in the ontogenesis including the development of the nervous system (brain development). Later on, the foetus is able to synthesise thyroid hormones in late gestation by his own, but it still needs a hormone delivery from the maternal side. The transport systems for iodine are extremely effective. In case of high iodine intake (see selenium), the thyroid gland is not protected against a fatal iodine

uptake. The placenta also cannot prevent a detrimental uptake of iodine by the foetus and burdens the foetal thyroid tissue by elevated iodine uptake. The proliferation of the tissue and size enlargement is visible already in the new-born (Table 4) and associated with defects in connective tissues.

*Table 4. Consequences of iodine intake by the mare during late gestation and the incidence of goitre in new-born foals (Meyer, 1998).*

| J-intake of the mare (mg/day) | goiter, incidence | others                                                |
|-------------------------------|-------------------|-------------------------------------------------------|
| 6-7                           | 0                 |                                                       |
| 48-55                         | 3%                | weakness, depressed muscle development                |
| 56-69                         | 10%               | contracted tendons                                    |
| 100-300                       | 25%               | retarded growth of skeleton                           |
| 83                            | 33%               | disturbed leg conformation                            |
| 288-432                       | 50%               | long hair                                             |
| 375                           | 100%              | absorption, osteopetrosis, disturbed leg conformation |

## **Cobalt/Vitamin B<sub>12</sub>**

Most cobalt research is done in ruminants as cobalt is the central part of vitamin B<sub>12</sub> and associated with microbial digestion. Experimental based information about cobalt in horses are rare. From enforced renal excretion after injection of cyanocobalamin<sup>60</sup>Co into the caecum it is concluded that this trace element which is incorporated in cobalamin will be absorbed in the hindgut (Stillions *et al.*, 1971). In contrast to the other trace elements absorption of cobalt means the absorption of the cobalt-dependent product, the cobalamin. Specialized plasma protein transports the cobalt-complex in the blood. The cobalamin concentrations in plasma and liver of ruminants increase in dependence on intake (Stemme *et al.*, 2002), and a drop in intake result in rapid changes in tissue cobalamin. Therefore a constant replenishment of cobalt for cobalamin synthesis is required. It is questionable if the utilisation rate of cobalt for cobalamin synthesis from ruminants (~ 37-65 % in dependence on cobalt intake) can be used for the equine. But the microbial synthesis of cobalamin by the equine intestinal community occurs and obviously serves sufficient cobalamin to the organism (Salminen, 1975). Hindgut fermenters seem to store no cobalamin in the liver as there is nearly an equilibrium between plasma and liver in animals with natural cobalt intake (McOrist and Thomas, 1984).

Cobalt and cobalamin respectively take part in energy metabolism by methylcobalamin as a donor of methyl groups. So far it is involved in elongation of carbon chains. It also activates other enzymes as donors of methyl groups, an important link to steroid metabolism. Adenosylcobalamin is a second cobalt depending complex which operates in the formation of glucose from volatile fatty acids. That is a key function in energy metabolism. The depression of  $\beta$ -oxidation of mobilised fatty acids in case of a shortage in adenosylcobalamin demonstrates the role of cobalt and related compound for the entire energy metabolism and liver function.

Cobalt intake by the gravid mammal determines the cobalamin status of the newborn and the cobalamin supply to the young by milk (Stemme *et al.*, 2002). In addition, the cobalamin

supply to the embryo and the foetus depend on maternal cobalamin status (no synthesis of cobalamin but remarkable toxicity of cobalt for embryo and foetus, Szakmáry *et al.*, 2001).

## Vitamins

The „Vita“ including term vitamins was chosen to express the importance of indispensable organic substances for physiological functions in any tissue and metabolic reactions even at low doses. That of course implies the contribution of vitamins to the processes of growth. Some aspects are already mentioned and the following comments are focussed on the main points.

It is described that vitamin A (retinol) is required for the implantation, early stages of embryonic life and throughout the organogenesis as well as for the integrity of epithelia (Schweigert, 2000). Retinol crosses the placenta rather independent of maternal intake. Experimental studies e.g. in rats demonstrate the sensitivity of fetal tissue development (like in lung and heart) against an insufficient maternal retinol intake (Antipatis *et al.*, 2000). The liver is the main organ to store vitamin A (Landes, 1994) and enables the organism to compensate for high or low intake. Reproduction failure as well as depressed vitality of the newborn are consequences of a vitamin A deficit (Meyer, 1998). The risk for the growing vitamin A-depleted animal is a result of the keratinisation of epithelial cells in several tissues including GIT, lung etc.

Vitamin D (1,25-dihydroxy-cholecalciferol) is a part of the mineral regulation and of special concern for bone growth. The precise role of vitamin D in the equine species is not sufficiently investigated as some results (experiments with low number of animals) indicate a low requirement (Breidenbach *et al.*, 1998). But this needs confirmation by further experiments. However, in principle there is no doubt that in experimental induced vitamin D depletion in horses bone deformations occur similar to those which are well known from other species (El Shorafa *et al.*, 1979).

Vitamin E represents a part of the antioxydative capacity in the organism. Particular the integrity of membranes are sensitive for vitamin E. The liver is an important storage side for tocopheroles, but also other tissues like lung responds to vitamin E intake. The growing foal depends on the mare's vitamin E status as the milk is the only vitamin E source for the foal during the first weeks of life. The mean impact on growth is less attributed to specific processes of growth but more related to the influence on immune status.

Vitamin K normally is understood as an essential factor for blood clotting. For growth its role in bone formation may be more important and often overlooked. The function of osteocalcin in the mineralization process in bone and the availability of matrix proteins in bone and of growth factors are vitamin K-dependent (Gundberg and Nishimoto, 1999). Vitamin K is synthesized in plants and by the intestinal micro flora. But the role of vitamin K of microbial origin is – excepted in ruminants – not investigated in the horse. Negative clinical experiences in long lasting use of antibiotics on clotting properties of blood indicate an well defined role of the intestinal synthesis.

Some results indicate a specific requirement for folic acid during the embryonic phase. Folate is involved in transformation of serin to glycine, purine synthesis, DNA-synthesis, formation of methionine from homocysteine and formation of glutamate (Barkow *et al.*, 1999). Particular embryonic survival rate seems to be improved after folate supplementation (Barkow *et al.*,

1999). The results are focussed on pig and not confirmed in ruminants (Flachowsky, 1999). Research is requested to investigate folate in mares particular concerning congenital defects (Toribio, 1999; Piercy *et al.*, 2002) after sulfonamide treatment.

## Conclusion

Selected aspects of nutrients are considered for reproduction and growth in the equine species. The role of macro- and trace elements as well as of vitamins is to recognize for all stages of growth. It should be underlined that the intrauterine phases of growth are included and particular these period is highly sensitive against changes in nutrient supply. The macro elements calcium, phosphorus and magnesium stand for bone formation. That is correct on one hand. But in addition the interaction with the hormonal control is important. Changing the supply in these elements induces a hormone response. The electrolytes take place by their importance for water and acid-base balance.

The trace elements participate in growth as “co-workers” in many metabolic reactions. These are important for bone, tendons, muscle, immune system etc. Vitamins represent a further group of micronutrients which interfere with reproduction and growth.

Several experiments and studies under the condition of feeding practice substantiate the importance of these nutrients for the young. The intensive interaction of these nutritional factor with processes of growth could stimulate a non deliberated use particular of trace elements and vitamins with consequences as fatal as the deficiency. Our recommendations need to be strongly based on facts regarding the physiological role of these nutrients.

Not in any cases the present knowledge is served by sufficient work in horses. Numerous questions are still waiting for scientific research to improve the quality of our skills in horse nutrition. That is more as a question of curiosity. The use of non proofed feed additives, non save feeding regimes, useless substances, toxic products is widely distributed in horse feeding. That condition is totally unacceptable.

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# Evaluation of mineral and vitamin requirements for growing horses

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## Introduction

Requirement figures are estimates based on available data. Depending on how the figures are defined, how they are derived, and what purpose they are intended for, there may be considerable differences between requirement figures. Users of requirement figures should keep this fact in mind and should avoid treating requirement figures as natural constants. It is highly recommended to read the background information which usually comes with tables on requirements before making an important decision or judgement based on a requirement figure.

## Definitions

- Net metabolic need, net requirements, requirements of available nutrient:  
These terms signify that the availability of the nutrient is not yet taken into account. For instance, the net metabolic need for calcium would be the accretion of calcium in bone and other tissue. Unless the availability is practically 100 % the intake of the amount that represents the net requirement of a nutrient for a given physiological state is not likely to support this physiological state.
- Minimum requirements:  
The minimum requirement represents the minimal amount of a maximally bioavailable nutrient that will support a given physiological state. Again the intake of the amount of a nutrient representing the minimum requirement may not support this physiological state under practical conditions (NRC, 2003).
- Recommended intake, recommended allowance, gross requirements:  
These terms refer to requirement figures which include a safety margin allowing for normal variability of bioavailability within the population and also within typical feed ingredients. Recommended intake of a nutrient should support a given physiological state under practical conditions (NRC, 2003).
- Adequate intake:  
Adequate intake is defined as the minimum amount of a nutrient demonstrated to support a defined physiological state when no minimum requirement is known (NRC, 2003).

## Derivement of requirement figures

There are several ways of arriving at requirement figures. The most common methods are i) factorial calculation and ii) dose response curves.

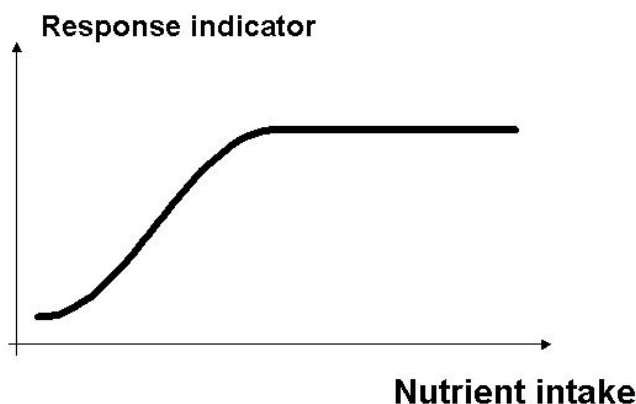
### *Factorial calculation*

For factorial calculation endogenous losses (faeces, urine, skin), losses by products (milk, foetus, placenta) and accretion of a nutrient in tissues are summed up as the net requirement. Then an estimate of bioavailability of the nutrient is made. In general a safety margin with

regard to variability of bioavailability under practical conditions is taken into account. Net requirements are divided by estimated bioavailability (in %) and multiplied with 100 to calculate the gross requirements or recommended allowance. Being the denominator of the calculation, the impact of bioavailability, an estimated variable, on the recommended allowance is enormous. Factorial calculation only works when the concentration of a nutrient in products or tissues is not affected by the amount of nutrient intake, except in a state of deficiency. Factorial calculation is mostly suitable for major minerals, and to a certain extent for some trace elements, but it cannot be recommended for deriving vitamin requirements. Results of factorial calculations are usually given per animal or on a body weight basis.

### *Dose response curves*

Figure 1 shows a typical dose response curve. A response indicator for the respective nutrient can be an enzyme to which the nutrient acts as co-enzyme, or the amount or activity of cells with a special association to the nutrient, or simply the concentration of the nutrient in a tissue. An example would be hematocrite as a response indicator for iron dose. In a rather crude version of dose-response experiments, the response indicator is the absence of clinical signs of deficiency. Especially in the older literature, this type of dose-response experiment is often done with two doses only: The lower dose induced clinical deficiency and the higher dose did not. The requirements are likely to be somewhere in between.



*Figure 1. Ideal dose-response curve.*

A problem with dose response curves is that the choice of the response indicators considerably affects the outcome of the experiment. The vitamin E-requirements for maximal activity of various immune cells of several species are considerably higher than the vitamin E-requirements for absence of clinical signs of deficiency (Senger, 2004). Dose response experiments do not completely avoid the problem of bioavailability. The result of a dose response experiment is only valid for similar conditions. For instance, figures obtained in animals on a semipurified diet with maximal availability of the respective nutrient cannot be transferred to practical diets without the addition of a safety margin which accounts for differences in availability. Nevertheless, dose response experiments are the method of choice to determine the requirements for vitamins and many trace elements. Results of dose response experiments are often given as concentration in the diet.

## Format of requirement figures

As described above, requirement figures can be given per animal, on a (metabolic) body weight basis, or as concentration in the diet, either on dry matter or on an energy basis. Transformation from one format to another requires knowledge on energy intake, and in case of figures given on a dry matter basis, also of energy density of the food. If these variables are not taken into account, transformations from one format to the other can lead to considerable errors.

Since it is not possible to review the requirements of all minerals and vitamins within the requested time frame, examples are chosen.

## Calcium and phosphorus requirements for growth

Calcium and phosphorous requirements for growth have been factorially derived by the Society of Nutrition Physiology (SNP, 1982 and 1994) and the NRC (1989). Both groups came to the result that a foal with an expected mature weight of 600 kg needs about 40 g calcium per day at the beginning of the growth and that this requirement decreases gradually to about 30 g per day at the end of the growth period. These figures include calcium for accretion in tissue, especially bones, and endogenous losses. They presume an average growth rate. The figures are calculated with a bioavailability of about 50 or 60 %, which can be considered to represent a reasonable safety margin, unless the calcium/phosphorous ratio is extremely narrow (<1:1) or the diet contains excessive amounts of phytate or oxalate. Therefore these figures can be regarded as a recommended intake, which should be adequate for balanced practical rations. The minimum requirements (presumed maximal availability of calcium) would then be about half to two thirds of the recommended intake. They may be even lower if the energy intake is also rather low and the foals have a below average growth rate. In a field study with 149 foals (Westfalian warm blooded horses, grazing pastures, often not supplemented, energy intake often below requirements) the intake of calcium did not meet the recommended intake (Finkler-Schade *et al.*, 1996a). In 26 % of the foals the calcium intake was even below 30 % of recommended intake. There were some occasional limb deviations in the foals from this study (Finkler-Schade *et al.*, 1996b); however, there were no obvious clinical signs of calcium deficiency, and the authors did not describe an association between limb deviations and calcium intake. Similar results were obtained in a recent study in Hanoverian warm blooded horses (Coenen, personal communication). This indicates that the minimum requirement may be sufficient to rear horses under extensive conditions in the field.

On the other hand, two field studies in thoroughbred foals and yearlings, one from Kentucky and the other from France, demonstrated that the incidence of developmental orthopedic disease (DOPD) is negatively correlated to calcium supply (Knight *et al.*, 1985; Paragon *et al.*, 2002). In both studies, there was a wide variety of calcium intake with the upper range being far above recommended intake. In the French study, it was also reported that a Ca/P ratio wider than 2:1 was associated with a lower incidence of DOPD.

A simple explanation for this effect might be that in breeding farms with high calcium supply to growing horses the awareness to DOPD was increased and/or the general management was superior, thus eliminating other factors contributing to the disease. There may be differences in the calcium content of the gain during growth and/or between breeds ("leggy" thoroughbred foals), which may lead to a underestimation of the factorially calculated

requirements. This, however, is not very likely, since the original data come from a light breed of horses and were obtained for 4 months old foals, yearlings and two-year-olds (Schryver *et al.* 1974). It has been suggested that a high calcium intake with a wide calcium/phosphorous ratio decreases bone turn-over in horses (Whitlock *et al.*, 1970; Lensing, 1998). A change in bone turn-over with or without alterations in the ratio of bone formation and bone break down could affect the incidence of DOPD in predisposed horses. In certain breeds of dogs, mainly Great Danes, such an effect has been reported (Hazewinkel *et al.*, 1985); however, in this species a high calcium intake increased the incidence of osteochondrosis. It is highly remarkable that a high calcium intake and a possible decrease in bone turn-over appears to affect DOPD in horses in the opposite way as in Great Danes. Explanations can so far only be speculative. Several findings suggest that there are major species differences in the calcium metabolism of horses and dogs. In contrast to dogs horses excrete major percentages of dietary calcium by urine (Meyer and Coenen, 2002). Vitamin D and its metabolites may not play an essential role in calcium homeostasis in the horse (Meyer and Coenen, 2002). This might affect the effects of parathyroid hormone (PTH) on calcium absorption. The horse is much more sensitive to an inverse calcium/phosphorous ratio than the dog or cat (NRC, 1989; Meyer and Zentek, 2001; Kienzle *et al.*, 1998), whereas the latter are more sensitive to a wide calcium/phosphorous ratio. Jordan *et al.* (1975) reported that the proportions of demonstrable polypeptide producing chief cells in the parathyroid of ponies eating a high calcium diet were approximately twice those of ponies fed conventional calcium levels. Lensing (1998) reported an increased of PTH-fragments in ponies eating a high calcium diet, whereas in dogs under the same conditions PTH decreases (Schoenmakers *et al.*, 1999).

In order to obtain a calcium/phosphorous ratio greater than 2:1 in a ration with an adequate phosphorous supply, the calcium intake needs to be up to 30 % above the factorially calculated figures for recommended dietary allowance. So far there are no reports of detrimental effects of high calcium intake (up to 5 times requirements) in growing horses, provided the intake of phosphorous is adequate (NRC, 1989; Meyer and Coenen, 2002). Jordan *et al.* (1975), however, reported some effects of a calcium/phosphorus ratio of 6 :1 on the relative size of medullary region (larger) and cortical bone area (smaller) in ponies. These changes were not accompanied by any clinical signs of stiffness or lameness. Nevertheless, it may be prudent not to increase the calcium intake and especially the calcium/phosphorous ratio to such an extent.

For phosphorous the factorially calculated requirements are about 20 g in a growing horse with an expected mature weight of 600 kg (SNP, 1982, 1994; NRC, 1989). The changes during the growth period are less marked than for calcium. The presumed bioavailability of around 40 % for the factorial calculation takes into account that rations for horses may contain considerable amounts of phytate, which may decrease phosphorous absorption. Therefore, the factorially calculated phosphorous requirements can be considered to represent a recommended dietary allowance. This is in agreement with the results of the above mentioned field studies (Finkler-Schade *et al.*, 1996a and b), where foals did not receive the recommended allowance of phosphorous without developing clinical signs of deficiency. During the 4<sup>th</sup> month of life the phosphorous intake of all foals was between 40 and 60 % below recommended dietary allowance. Later the majority of the foals received between 10 % and 40 % less phosphorous than recommended. Therefore it appears likely that the minimum requirement of phosphorous is about half the recommended dietary allowance. Whereas phosphorous deficiency is very rarely observed in horses, excess of dietary phosphorous combined with an inverse calcium/phosphorous ratio and/or calcium deficiency are long since

known to have detrimental effects on skeletal health of horses (NRC, 1989). At the moment there is no indication that it may be necessary to revise the phosphorous requirements (recommended dietary allowance) for growth.

## **Copper requirements for growth**

Copper requirements for growth have been estimated to amount to 10 mg/kg dry matter by several groups (Jarridge and Martin-Rosset, 1981; SNP, 1982; SNP, 1994; NRC, 1989; Meyer, 1994). The methods included both, factorial calculation and experiments with satisfactory growth as response indicator. Bridges and Harris (1988) demonstrated that for the first few months of life 1.7 mg Cu/kg dry matter is not enough. Hurtig *et al.* (1991) demonstrated clinical and gross morphologic signs of copper deficiency in foals weaned with three months of age on a diet of 7 mg Cu/kg dry matter, whereas 30 mg/kg dry matter supported growth but induced high liver copper contents. Knight *et al.* (1990) found that 15 mg Cu/kg dry matter in additional feed for sucking foals were not sufficient to prevent chondrotic lesions. On the other hand Finkler-Schade *et al.* (1996a and b) found that practically all sucking foals kept on pasture with their mothers in Westfalia had less than 10 mg Cu/kg dry matter in their rations. There were no clinical signs of copper deficiency. Paragon *et al.* (2002) did not find differences in the incidence of DOPD in relation to copper intake. In this study copper requirements of 10 mg/kg dry matter were met or exceeded. By contrast in the field studies of Knight *et al.* (1985) a high incidence of DOPD was associated with lower copper intake.

At the moment it appears prudent to increase copper content in additional feeds for sucking foals to 20-25 mg/kg dry matter (Meyer, 1994), in order to increase the copper content in their diet to a total of at least 10 mg/kg dry matter. Judging from the few data on liver copper stores in newborn foals and analogous to other species, the copper intake of the pregnant mare is likely to influence the liver copper stores of the newborn foal (Meyer, 1994). Appropriate liver copper stores may be important for the foal to bridge the gap in copper supply during the first two to three months of life when the percentage of mares' milk in the ration of the foal is still high. In foals born with low copper stores and growing fast, it may be useful to feed copper supplements or even apply copper solutions intravenously. This has a long tradition. The so-called "Trakehner Kupfer Lösung" was injected when foals showed anaemia or developmental diseases (Meyer, 1994).

## **Zinc requirements for growth**

This element provides a good example for a dose-response curve with only two dots on it. Harrington *et al.* (1973) fed foals a purified diet with either 5 or 40 mg Zn/kg dry matter. The foals eating 5 mg Zn/kg dry matter developed clinical signs of deficiency whereas the other group grew normally. Feed stuffs for horses contain rather often less zinc than 40 mg/kg dry matter; however, zinc deficiency is an extremely rare problem in foals. Therefore it is quite likely that lower zinc levels than 40 mg/kg dry matter may support growth. As with other minerals Finkler-Schade *et al.* (1996a and b) reported that a majority of Westfalian foals ate less zinc than recommended without developing clinical signs of deficiency. The requirement of 40 mg/kg dry matter appears to include a safety margin, and would represent rather a recommended dietary allowance than a minimum requirement. By contrast, Knight *et al.* (1985) reported a negative correlation between the zinc concentration and the perceived degree of affliction with metabolic bone disease in weanlings in a field study. In this study, however, there was also a similar negative correlation between calcium supply and copper

supply and metabolic bone disease. It is most likely that calcium, copper and zinc supply are positively correlated, simply because they usually are fed in mineral mixtures. Therefore it is not clear which of these three minerals was most important for bone development.

## **Vitamin A requirements for growth**

A number of different substances possess biological vitamin A activity or can be biologically transformed into an active vitamin A. In horses (as in many other species) most experiments to determine vitamin A requirements have been performed with retinol and its esters. Requirements for growth are estimated to amount to 150-200 IU/kg body weight or 2000 IU/kg dry matter. These figures were derived from a curvilinear dose response curve with four dots: Mild deficiency, controls, mild intoxication and intoxication (low carotene and no retinol, 35 IU/kg body weight, 3500 IU/kg BW and 35000 IU/kg BW, respectively). The intake of the controls was supposed to be suboptimal from the dose-response curves using growth and hematologic and biochemical parameters (Donoghue *et al.* 1981). Although it has been demonstrated that vitamin A plays a role in bone growth and remodelling in other species, so far this role of vitamin A has not been clearly demonstrated in horses (Senger, 2004). On the other hand Donoghue *et al.* (1981) demonstrated that vitamin A intoxication in growing foals may induce unthriftiness, ataxia, and hyperextension of carpal, tarsal and metapalangeal joints. The latter might be due to poor muscle tonus, while ataxia could be related to disorders of the development of the spine or head bones. Lensing (1998) demonstrated that even adult ponies responded to excess vitamin A (1000 IU/kg body weight/d) over 30 days with a reduction of various bone markers for both resorption and production of bone tissue, i.e. with reduced bone remodelling activity. From the view point of preventing DOPD in fast growing foals from large breeds, therefore, vitamin A excess may be considered to be more critical than vitamin A deficiency. This is also the case from the view point of feeding practice: Foals and their mothers usually are at least part time grazing on pastures, which contain bioavailable carotenes that can be transformed to vitamin A. This makes vitamin A deficiency very unlikely. By contrast, foals given additional feed with added vitamins and one or more vitamin supplements may eat much more biologically active vitamin A than they need. In this context, it is most important to note that while retinol is inevitably biologically active, carotenes are not. They are only transformed into the active retinol form if there is a need for it. Therefore, excessive carotene intake (such as grazing lush pastures or eating lots of carrots) does not lead to vitamin A intoxication (NRC, 1989). For ration calculation it is recommended therefore to differentiate between carotene and retinol intake.

## **Vitamin D requirements for growth**

The biological function of vitamin D in horses appears to differ from the functions usually associated to this vitamin. In horses even overdoses of vitamin D appear not to affect plasma calcium content (Breidenbach *et al.*, 1998). By contrast plasma phosphate increased considerably. Apparent digestibility of phosphorus increased whereas apparent calcium digestibility tended to decline. Plasma levels of 25OHD<sub>3</sub> and 1,25OHD<sub>3</sub> are lower than in other mammals. Intramuscular injection of vitamin D<sub>3</sub> increased vitamin D<sub>3</sub> levels in plasma but 25OHD<sub>3</sub> and 1,25OHD<sub>3</sub> were not affected (Harmeyer *et al.*, 1992). El Shorafa *et al.* (1979) postulated a need for vitamin D in growing ponies kept in darkness but not in growing ponies kept in daylight in Florida. They did not state the level of calcium and phosphorous in their diets. Judging from the ingredients, it appears likely that the levels of both minerals were

marginal. Vitamin D may have enhanced the utilisation of either mineral, most likely of phosphorous.

For growing horses with an appropriate supply of calcium and phosphorous and exposition to sunlight vitamin D is unlikely to be essential. If there is a requirement for vitamin D, it is again unlikely to exceed the content of sun cured hay of about 500 IU/kg air dry material (Meyer and Coenen, 2002). On the other hand vitamin D is relatively toxic for horses. Although overdoses of vitamin D do not induce hypercalcemia in horses, they do induce major soft tissue calcification. Weisweiler *et al.* (1993) injected 10,000 IU/kg body weight on four consecutive days in adult ponies and observed marked symptoms of toxicity including massive soft tissue calcification, hyperphosphatemia and tachycardia. In summary, it is not recommended to use vitamin D as an additive in mixed feed in concentrations that will lead to vitamin D concentrations in the total ration which considerably exceed the vitamin D content of sun cured hay.

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## Common feeding practices and supply of minerals and vitamins

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## Introduction

A balanced ration for growing horses is commonly composed of roughages, concentrates and premixes and must provide the horses all the nutrients they need for a sound growth.

To achieve this result, it is important to know very well the horse's requirements from one hand, and the feedstuffs and premixes nutritional value and supply from the other, in order to estimate the unbalances that can arise from the use of different rations.

As to minerals and vitamins, there is a huge variety of situations: for roughages, and in particular for hays, for example, the mineral content of the final product depends on the grass type, the soil composition, the harvesting conditions, the grass growing stage at cutting, the storage conditions, and for this reason it is important to give some figures of the roughages "most commonly" used for horse's feeding in Europe. Variability is also a peculiarity of the mineral and vitamin content of simple feeds.

As to the compound concentrates, these products are commonly added premixes and present a huge variety of vitamins and minerals levels, according to the quantity that is to be administered per day and to the roughage supposed to complete the ration.

In this paper, we will give and comment some figures on the mineral and vitamins composition of different feeds and premixes used in Europe, and to point out the most common problems of practical rationing.

## Mineral and vitamin requirements for growing horses

There are several sources of data providing figures about the mineral and vitamin requirements of growing horses. Here are reported some tables (Table 1-5) from INRA, NRC and Wolter. Some data about toxic levels are also reported from the last Author.

*Table 1. mineral requirements (g/day) of growing horses of mature weight 450 kg (INRA, 1990).*

| Age (months) and growth rate; mature weight 450 kg | Ca | P  | Mg | Na | DM intake |
|----------------------------------------------------|----|----|----|----|-----------|
| 8-12, optimal                                      | 36 | 20 | 9  | 11 | 5.0-7.5   |
| 8-12, mild                                         | 25 | 14 | 9  | 9  | 4.5-7.0   |
| 20-24, optimal                                     | 33 | 19 | 9  | 12 | 7.0-10.0  |
| 20-24, mild                                        | 24 | 14 | 8  | 10 | 6.5-9.5   |
| 32-36, optimal                                     | 26 | 15 | 9  | 11 | 7.7-10.0  |
| 32-36, mild                                        | 23 | 14 | 8  | 11 | 7.0-9.5   |
| Age (months) and growth rate; mature weight 600 kg |    |    |    |    |           |
| 8-12, optimal                                      | 44 | 24 | 12 | 13 | 6.5-9.0   |
| 8-12, mild                                         | 33 | 18 | 11 | 11 | 6.0-8.0   |
| 20-24, optimal                                     | 42 | 24 | 10 | 15 | 8.5-12.0  |
| 20-24, mild                                        | 33 | 19 | 10 | 13 | 8.0-11.0  |
| 32-36, optimal                                     | 37 | 22 | 10 | 15 | 9.0-13.0  |
| 32-36, mild                                        | 29 | 17 | 11 | 14 | 9.5-12.0  |

*Table 2. Mineral requirements for growing horses of mature weight 500 kg (g/day), a comparison between INRA and NRC tables (Wolter, 1994, modified).*

| Age (months) | Ca NRC | Ca INRA | P NRC | P INRA | Ca/P NRC | Ca/P INRA | DM intake INRA |
|--------------|--------|---------|-------|--------|----------|-----------|----------------|
| 6            | 36     |         | 20    |        | 1.8      |           |                |
| 12           | 34     | 39      | 19    | 22     | 1.8      | 1.8       | 5.5-8.0        |
| 24           | 34     | 36      | 19    | 20     | 1.8      | 1.8       | 8.0-11.0       |

*Table 3. Trace elements recommended allowances and toxicity levels (mg/kg DM in the diet), (Wolter, 1994, modified).*

|           | Requirements (NRC, 1989) | Recommended allowances according to Wolter (1994) | Toxicity threshold (NRC, 1989) |
|-----------|--------------------------|---------------------------------------------------|--------------------------------|
| Iron      | 50                       | 100                                               | 1,000                          |
| Copper    | 10                       | 30                                                | 800                            |
| Zinc      | 40                       | 75                                                | 500-1,000                      |
| Manganese | 40                       | 50                                                | 1,000                          |
| Cobalt    | 0.1                      | 0.15                                              | 10                             |
| Selenium  | 0.1                      | 0.2                                               | ≥3                             |
| Iodine    | 0.1-0.6                  | 0.2                                               | ≥5                             |

*Table 4. Daily vitamin requirements of growing horses (Wolter, 1994, modified).*

| Age (months)           | 0-2   | 2-6    | 6-18   | Over 18 |
|------------------------|-------|--------|--------|---------|
| Vit. A (IU)            | 5,000 | 15,000 | 25,000 | 35,000  |
| Vit. D (IU)            | 750   | 1,500  | 3,000  | 6,000   |
| Vit. E (IU)            | 10    | 20     | 50     | 100     |
| Vit. B1 (mg)           | 3     | 6      | 12     | 24      |
| Vit. B2 (mg)           | 5     | 10     | 20     | 40      |
| Niacin (mg)            | 15    | 30     | 60     | 120     |
| Panthothenic acid (mg) | 6     | 12     | 24     | 48      |
| Vit. B6 (mg)           | 1.5   | 3      | 6      | 12      |
| Choline (mg)           | 75    | 150    | 300    | 600     |
| Folic acid (mg)        | 1.5   | 3      | 6      | 12      |
| Vit. B12 (mg)          | 15    | 30     | 60     | 120     |

*Table 5. Vitamin requirements of growing horses per kg of DM ingested (NRC).*

|              |       |
|--------------|-------|
| Vit. A (IU)  | 2,000 |
| Vit. D (IU)  | 800   |
| Vit. E (IU)  | 80    |
| Vit. B1 (mg) | 3     |
| Vit. B2 (mg) | 2     |

## Vitamin and mineral content of roughages

As to roughages for growing horses, the most commonly used in Europe are hays and, to a lesser extent, pasture grass. The use of haylages is still less common. Some data on mineral content of hays are provided by the French tables published by INRA (1990). The following selection is referred to different products chosen for their availability and common use in breeding farms.

*Table 6. Mineral content of different hays and pasture grasses, g/kg DM (INRA, 1990).*

|                                                                   | Ca   | P   | Mg  |
|-------------------------------------------------------------------|------|-----|-----|
| Permanent pasture hay, first cut, earing, first quality           | 6.0  | 3.0 | 2.0 |
| Permanent pasture hay, first cut, flowering, first quality        | 5.5  | 2.5 | 1.5 |
| Permanent pasture hay, first cut, flowering, poor quality         | 4.5  | 2.0 | 1.0 |
| Permanent pasture hay, first cut, end of flowering, first quality | 5.5  | 2.0 | 1.5 |
| Permanent pasture hay, first cut, flowering, poor quality         | 4.5  | 1.5 | 1.0 |
| Alfalfa hay, first cut, flowering                                 | 15.0 | 2.0 | 1.5 |
| Alfalfa hay, second cut                                           | 15.0 | 2.0 | 1.5 |
| Permanent pasture, first cycle, 10 cm ear                         | 7.5  | 4.5 | 2.0 |
| Permanent pasture, first cycle, earing                            | 6.0  | 3.0 | 2.0 |
| Permanent pasture, second cycle, 5 weeks after cut                | 8.0  | 4.0 |     |
| Permanent pasture, second cycle, 8 weeks after cut                | 7.5  | 3.5 | 2.5 |
| Permanent pasture, third cycle, 6 weeks                           | 6.5  | 4.0 | 3.0 |
| Alfalfa, first cycle, sprouting                                   | 17.0 | 3.0 | 2.0 |
| Alfalfa, second cycle, 5 weeks                                    | 16.0 | 3.0 | 2.5 |

The same source provides some figures on the trace elements contained in hays and grasses.

*Table 7. Trace elements content of different pasture grasses and hays, mean values, mg/kg DM (INRA, 1990, modified).*

|                                           | Cu  | Zn   | Mn    | Mo   |
|-------------------------------------------|-----|------|-------|------|
| Permanent pasture, first cycle, grazed    | 7.4 | 48.0 | 149.0 | 0.87 |
| Permanent pasture, first cycle, earing    | 5.9 | 36.0 | 148.0 | 0.83 |
| Permanent pasture, first cycle, flowering | 5.0 | 34.0 | 141.0 | 0.75 |
| Ryegrass, first cycle, earing             | 4.0 | 19.5 | 74.0  | 1.01 |
| Ryegrass, second cycle                    | 5.2 | 26.5 | 148.4 |      |
| Alfalfa, first cycle, sprouting           | 7.5 | 22.9 | 25.5  | 1.07 |
| Alfalfa second cycle                      | 8.5 | 22.3 | 52.7  | 0.47 |
| Permanent pasture hay, first cut          | 5.2 | 29.1 | 158.2 | 0.63 |
| Italian ryegrass hay, first cycle         | 4.9 | 26.5 | 110.0 |      |
| Alfalfa hay, first cut                    | 7.1 | 24.6 | 29.0  |      |
| Alfalfa hay, second cut                   | 7.5 | 23.7 |       |      |

As it is clear, the type of roughage used can deeply change the “basal” mineral value of the ration for a growing horse. This must be taken in due account in the evaluation of the mineral and vitamin value of different rations that doesn’t include vitamin and mineral premixes.

Some more data are also available from two different studies carried out in Norway: in the following two tables some figures about the mineral content range are provided.

*Table 8. Mineral content of hay in Norway (n=70); (Austbø, personal communication).*

| Mineral       | Mean  | Minimum | Maximum            | St dev |
|---------------|-------|---------|--------------------|--------|
| Percent of DM |       |         |                    |        |
| Ca            | 0.304 | 0.115   | 0.876 <sup>1</sup> | 0.124  |
| P             | 0.208 | 0.066   | 0.359              | 0.051  |
| Mg            | 0.117 | 0.005   | 0.304              | 0.038  |
| K             | 1.846 | 0.814   | 2.781              | 0.465  |
| Na            | 0.040 | 0.007   | 0.238              | 0.048  |
| Mg/kg DM      |       |         |                    |        |
| Fe            | 103.2 | 38.0    | 422.5              | 84.9   |
| Cu            | 6.8   | 3.1     | 15.7               | 2.4    |
| Mn            | 72.5  | 29.2    | 259.7              | 37.9   |
| Zn            | 23.7  | 10.8    | 46.5               | 6.5    |
| Mo            | 0.57  | 0       | 1.8                | 0.5    |
| Al            | 71.4  | 11.1    | 256                | 71.3   |

<sup>1</sup> Hay with a high content of clover, not very common in Norway.

Some studies carried out in Germany refer to the mineral composition of different roughages, including silages, used for horses. The following table gives the main figures of these studies.

Table 9. Mineral content of hay in Norway (n=55); (Austbø, personal communication).

| Mineral       | Mean  | Minimum | Maximum | St dev |
|---------------|-------|---------|---------|--------|
| Percent of DM |       |         |         |        |
| Ca            | 0.317 | 0.132   | 0.586   | 0.115  |
| P             | 0.243 | 0.108   | 0.383   | 0.051  |
| Mg            | 0.134 | 0.058   | 0.313   | 0.051  |
| Mg/kg DM      |       |         |         |        |
| Cu            | 5.54  | 1.9     | 13.4    | 2.36   |
| Zn            | 22.16 | 10.0    | 42.8    | 6.88   |

Table 10. Minerals in grass, grass silages and hay (Borchers, 2002; Granel, 2002; Müller, 2002; Hoogeback, unpublished data).

|              | N   | Average | Upper level | Lower level |
|--------------|-----|---------|-------------|-------------|
| Ca, g/kg DM  | 559 | 4.6     | 13.2        | 1.3         |
| P, g/kg DM   | 560 | 3.6     | 9.4         | 2.0         |
| Cu, mg/kg DM | 561 | 8       | 31          | 1           |
| Zn, mg/kg DM | 549 | 38      | 106         | 7           |
| Mn, mg/kg DM | 561 | 143     | 818         | 2           |
| Se, mg/kg DM | 41  | 0.05    | 0.17        | 0.00        |

## Vitamin and mineral content of concentrates

Basal rations for horses are, very often, still based on roughages and simple feedstuffs, and oats in particular. For this reason, it is important to assess the mineral and vitamin content of these products and to provide information about variability. We present here, as a sample of the available data, the mineral and vitamin content of 5 different common feeds widely used in horse rations: oats, barley, soft wheat coarse bran, horse beans and soybean meal. The data were obtained from the “Raw material compendium” (NOVUS, 1994), that is a compilation of feed composition from 17 different data sources including NRC, INRA, data from MAFF (UK), Feedstuffs, WPSA, Becker & Nehring and some other sources including databases from Asia, Latin America and Mid East.

Other studies confirm this variability. Austbø (personal communication) provided some interesting data on oats mineral composition, shown in the following table.

*Table 11. Minimum and maximum content of different minerals and vitamins in simple feeds for horses, as fed (Novus, 1994).*

|               | Oats      | Barley     | Soft wheat<br>coarse bran | Horse beans | Soybean meal<br>44% CP |
|---------------|-----------|------------|---------------------------|-------------|------------------------|
| Ca, %         | 0.07-0.20 | 0.05-0.15  | 0.04-0.24                 | 0.09-0.17   | 0.25-0.39              |
| P, %          | 0.26-0.37 | 0.32-0.41  | 1.00-1.30                 | 0.38-0.61   | 0.62-0.71              |
| Mg, %         | 0.08-0.26 | 0.10-0.15  | 0.27-0.55                 | 0.12-0.18   | 0.16-0.32              |
| Na, %         | 0.01-0.07 | 0.01-0.07  | 0.01-0.06                 | 0.01-0.80   | 0.01-0.39              |
| Fe, mg/kg     | 57-125    | 50-153     | 35.8-200                  | 74.82-82    | 104.9-367              |
| Cu, mg/kg     | 2.83-6    | 3.7-8      | 9-30                      | 4.10-13     | 14-22                  |
| Mn, mg/kg     | 37-63     | 14.5-26    | 37-132                    | 6.72-28.71  | 25.6-49                |
| Zn, mg/kg     | 22.4-37   | 17-30      | 74-162                    | 33.06-50    | 27.3-61.6              |
| Se, mg/kg     | 0.1-0.3   | 0.09-0.20  | 0.24-0.70                 | 0.02        | 0.10-0.60              |
| Co, mg/kg     | 0.02-0.23 | 0.01-0.09  | 0.05-0.10                 | 0.03-0.40   | 0.09-1.28              |
| I, mg/kg      | 0.09-0.17 | 0.04-0.35  | 0.07-2.10                 |             | 0.15-0.51              |
| Vit A, IU/kg  | 600       |            | 400                       |             |                        |
| Vit. E, IU/kg | 3.35-24   | 6.51-47    | 10-28                     | 0.7-9       | 0.67-3.5               |
| B1, mg/kg     | 5.1-7.0   | 4.0-4.8    | 6.0-8.5                   | 5.5         | 1.7-5.4                |
| B2, mg/kg     | 1.1-2.0   | 1.3-2.2    | 2.9-5.1                   | 1.6-3.6     | 2.88-4.0               |
| B6, mg/kg     | 1.2-2.0   | 2.9-7.5    | 6.0-25.0                  |             | 6.0-7.3                |
| Niacin, mg/kg | 13.2-22.0 | 41.0-74.94 | 177-321                   | 22.4-29.0   | 26.9-59.8              |
| Folate, mg/kg | 0.32-0.50 | 0.30-0.57  | 1.4-4.0                   |             | 0.45-3.63              |

*Table 12. Mineral content of oats sampled from horse owners 1990 (n=5) (Austbø, personal communication).*

| Mineral       | Mean  | Minimum | Maximum | St dev |
|---------------|-------|---------|---------|--------|
| Percent of DM |       |         |         |        |
| Ca            | 0.075 | 0.064   | 0.086   | 0.007  |
| P             | 0.346 | 0.271   | 0.370   | 0.038  |
| Mg            | 0.120 | 0.104   | 0.137   | 0.012  |
| K             | 0.377 | 0.320   | 0.482   | 0.064  |
| Na            | 0.005 | 0.004   | 0.007   | 0.001  |
| Mg/kg DM      |       |         |         |        |
| Fe            | 61.2  | 41.3    | 94.4    | 22.9   |
| Cu            | 6.2   | 4.3     | 12.5    | 3.1    |
| Mn            | 39.6  | 30.1    | 51.5    | 7.8    |
| Zn            | 31.6  | 22.1    | 34.4    | 7.8    |
| Mo            | <0.5  |         |         |        |
| Al            | 7.9   | 5.1     | 14.4    | 3.7    |

## Vitamin and mineral content of mixed feeds

When compound feeds are used for horse rationing, the situation is more complex: in fact supplements are added in different amounts, hence changing the final content both for minerals and vitamins. The specifications, provided by the manufacturers, stand for the vitamins and trace elements added with the premixes, and does not include the vitamins and trace elements content of the ingredients. The labels, then, give an approximate idea of the

real level of these nutrients in the feed. Below, the compositions of different compound feed for horses (breeding mares and foals) are reported together with the range.

*Table 13. Breeding mares compound feeds used in Germany. Data are expressed as fed, assuming 880g/kg DM; units are g/kg for Ca, P, Mg, Na, K, Cl; mg/kg for trace elements, B1, B6, B12, niacin, biotin and carotin; IU/kg for vitamins A, D and E (Coenen, personal communication).*

|         | Ca    | P     | Mg    | Na  | K   | Cl  | Cu     | Zn     | Se      | I   |
|---------|-------|-------|-------|-----|-----|-----|--------|--------|---------|-----|
| min     | 8     | 4     | 1.7   | 2   | 10  | 2   | 13     | 65     | 0.2     | 0.3 |
| max     | 24    | 12    | 6.5   | 5   | 13  | 3   | 90     | 800    | 2       | 8.5 |
| average | 13    | 6     | 2.9   | 3.5 | 11  | 3   | 35     | 217    | 0.64    | 1.9 |
| n       | 17    | 17    | 14    | 14  | 3   | 2   | 16     | 13     | 14      | 12  |
|         | Vit.A | Vit.D | Vit.E | B1  | B6  | B12 | Niacin | Biotin | carotin |     |
| min     | 12    | 1.25  | 0.08  | 2   | 0.4 | 20  | 15     | 0.1    | 4000    |     |
| max     | 100   | 8     | 5.5   | 50  | 40  | 120 | 120    | 2.4    | 4000    |     |
| average | 31.8  | 2.91  | 0.53  | 12  | 11  | 53  | 51.17  | 0.7    | 4000    |     |
| n       | 18    | 18    | 18    | 11  | 10  | 10  | 9      | 10     | 1       |     |

*Table 14. Foals compound feeds used in Germany. Data are expressed as fed, assuming 880g/kg DM; units are g/kg for Ca, P, Mg, Na, K, Cl; mg/kg for trace elements, B1, B6, B12, niacin, biotin and carotin; IU/kg for vitamins A, D and E (Coenen, personal communication).*

|         | Ca    | P     | Mg    | Na  | K  | Cl  | Cu     | Zn     | Se      | I   |
|---------|-------|-------|-------|-----|----|-----|--------|--------|---------|-----|
| min     | 8     | 0.6   | 1.5   | 0.4 | 5  | 4   | 13     | 65     | 0.2     | 0.3 |
| max     | 27    | 13    | 4     | 6   | 10 | 9   | 75     | 560    | 1.4     | 1.5 |
| average | 13.8  | 5.9   | 2.4   | 3.1 | 7  | 5   | 33     | 157    | 0.55    | 1   |
| n       | 19    | 19    | 10    | 15  | 4  | 4   | 20     | 13     | 16      | 10  |
|         | Vit.A | Vit.D | Vit.E | B1  | B6 | B12 | Niacin | Biotin | Carotin |     |
| min     | 0.043 | 0     | 0.07  | 2   | 1  | 25  | 3      | 0.1    | 9       |     |
| max     | 60    | 6     | 1     | 35  | 28 | 80  | 110    | 250    | 11      |     |
| average | 26.74 | 2.76  | 0.25  | 11  | 11 | 50  | 48.5   | 37.33  | 10      |     |
| n       | 21    | 21    | 21    | 9   | 7  | 8   | 6      | 10     | 2       |     |

The data in the following three Tables (15, 16 and 17), recommendations and comments are referred to concentrates used in Norway. The concentrates “Product 1” and “Product 4” are specially designed for growing horses.

*Table 15. Macro minerals in Norwegian concentrate mixtures for horses (g/kg as fed) (Austbø, personal communication).*

|           | Ca   | P   | Mg  | Na  | Ca/P |
|-----------|------|-----|-----|-----|------|
| Product 1 | 16.0 | 8.0 | 3.0 | 4.0 | 2.0  |
| Product 2 | 14.5 | 6.0 | 3.0 | 4.0 | 2.4  |
| Product 3 | 6.0  | 4.4 | 3.0 | 3.5 | 1.4  |
| Product 4 | 15.5 | 7.5 | 2.5 | 3.0 | 2.1  |
| Product 5 | 18.0 | 6.0 | 4.0 | 3.5 | 3.0  |
| Product 6 | 6.0  | 4.5 | 3.0 | 3.0 | 1.3  |

*Table 16. Trace elements added to Norwegian concentrate mixtures for horses (mg/kg as fed) (Austbø, personal communication).*

|           | Fe  | Cu | Mn  | Zn  | Se   | Co   | I    |
|-----------|-----|----|-----|-----|------|------|------|
| Product 1 | 300 | 50 | 100 | 150 | 0.50 | 0.3  | 1.0  |
| Product 2 | 300 | 50 | 100 | 150 | 0.7  | 0.3  | 1.0  |
| Product 3 | 225 | 37 | 75  | 112 | 0.38 | 0.22 | 0.75 |
| Product 4 | 180 | 36 | 60  | 102 | 0.48 | 0.36 | 0.6  |
| Product 5 | 300 | 60 | 100 | 170 | 0.8  | 0.6  | 1.0  |
| Product 6 | 150 | 30 | 50  | 85  | 0.4  | 0.3  | 0.5  |

*Table 17. Vitamins added to Norwegian concentrate mixtures for horses (IU/kg for vitamins A, D and E; mg/kg for the other vitamins, as fed) (Austbø, personal communication).*

|           | Vit A | Vit D | Vit E | B1 | B2   | B6   | B12  | Niacin | Folate |
|-----------|-------|-------|-------|----|------|------|------|--------|--------|
| Product 1 | 9000  | 1000  | 600   | 25 | 15   | 3    | 0.2  | 10     | 5      |
| Product 2 | 9000  | 1000  | 600   | 25 | 15   | 3    | 0.2  | 10     | 5      |
| Product 3 | 6700  | 820   | 450   | 19 | 11.2 | 2.25 | 0.15 | 7.5    | 3.75   |
| Product 4 | 7200  | 720   | 540   | 12 | 9    | 4.2  | 0.06 | 18     | 3      |
| Product 5 | 12000 | 1200  | 900   | 20 | 15   | 7    | 0.1  | 30     | 5      |
| Product 6 | 6000  | 600   | 450   | 10 | 7.5  | 3.5  | 0.05 | 15     | 2.5    |

## Recommendations for the use of these products

### *Product 1*

0.4 – 0.5 kg/100 kg adult body weight from age 6-7 months (gradually increasing first weeks after weaning). Should be given as the only concentrate in addition to ad libitum hay or haylage of good quality. Oats may be added when the young horses start training.



#### *Product 4*

0.4 kg/100 kg adult body weight from age 6-7 months (gradually increasing first weeks after weaning). Should be given as the only concentrate in addition to *ad lib* hay or haylage of good quality. Oats may be added when the young horses start training

#### *Product 2*

0.5 kg/100 kg body weight. The energy of the total ration should be balanced mainly with oats or barley. Hay: 1.0 – 1.5 kg /100 kg BW.

#### *Product 5*

0.3 kg/100 kg body weight. The energy of the total ration should be balanced mainly with oats or barley. Hay: 1.0 – 1.5 kg /100 kg BW.

#### *Products 3 and 6*

These products are meant as the only concentrate for hobby horses, but if the horses are being trained, the ration can be supplied with oats or barley.

When these recommendations are followed, there should be no need for extra vitamin or mineral supplement, except for common salt (NaCl). One problem is that horse owners still use other supplements in addition to the concentrate mixtures thus increasing the risks of excesses.

The latter situation is pointed out for Norway, but is common in many other countries, and in particular in Italy.

### **Vitamin and mineral premixes**

When roughages and simple feeds are used for growing horses rationing, it is common to add a premix to meet the vitamin and trace elements requirements. Some of these premixes provide also adequate amounts of Ca, P, Na and Mg, but this is not common in all countries; in Italy, e.g. there are a very few premixes for growing horses providing Ca or P in large amounts on the market.

Here are reported the compositions and/or daily recommended doses of different premixes from different countries. Once again, the differences are very large.

*Table 18. Mineral/vitamin composition of 4 premixes for growing horses used in Italy. The data are referred to the daily supply (recommended daily doses on first row).*

| Dose (g/day)       | 30    | 25     | 12    | 150   |
|--------------------|-------|--------|-------|-------|
| vit. A IU          | 36000 | 250000 | 24000 | 7500  |
| vit. D3 IU         | 3600  | 5000   | 2400  | 1500  |
| vit. E mg          | 420   | 500    | 0     | 37.5  |
| vit. K mg          | 0     | 300    | 6     | 2.25  |
| vit. C mg          | 600   | 850    | 6     | 37.5  |
| vit. B1 mg         | 12    | 167.5  | 24    | 4.5   |
| vit. B2 mg         | 6     | 82.5   | 18    | 7.5   |
| vit. PP mg         | 0     | 65     | 12    | 18.75 |
| Panhotenic acid mg |       | 65     | 18    | 7.5   |
| vit. B6 mg         | 6     | 125    | 8.4   | 4.5   |
| vit. B12 mg        | 1.2   | 0.5    | 0.04  | 0.02  |
| Folic acid mg      | 30    | 35     | 6     | 0.75  |
| Inositol mg        | 300   | 0      | 0     | 0     |
| vit. H mg          | 0     | 1      | 0.9   | 0.08  |
| Coline mg          | 1200  | 1675   | 210   |       |
| beta-carotin mg    |       | 0      | 0     | 0     |
| Nicotinic acid mg  |       | 0      | 0     | 0     |
| Zn mg              |       | 300    | 120   | 150   |
| Fe mg              |       | 200    | 72    | 75    |
| Cu mg              |       | 100    | 12    | 15    |
| Mn mg              |       | 157.5  | 18    | 112.5 |
| Co mg              |       | 1.68   | 0.24  | 0.23  |
| Se mg              |       | 0.25   | 0.24  | 0.15  |
| I mg               |       | 2.5    | 0.18  | 1.13  |

*Table 19. Vitamin and mineral supplements used in Norway, stated composition. Recommended use is 50–100 grams a day for a 500 kg horse (Austbø, personal communication).*

|               | Product 1 | Product 2 | Product 3 |
|---------------|-----------|-----------|-----------|
| Ca, g/kg      | 110       | 140       | 170       |
| P, g/kg       | 70        | 30        | 35        |
| Mg, g/kg      | 30        | 20        | 60        |
| Na, g/kg      | 50        | 25        | 40        |
| Fe, mg/kg     | 5000      | 5000      | 450       |
| Cu, mg/kg     | 900       | 1200      | 850       |
| Mn, mg/kg     | 4000      | 2000      | 1500      |
| Zn, mg/kg     | 4000      | 4500      | 2000      |
| Se, mg/kg     | 20        | 30        | 25        |
| Co, mg/kg     | 10        | 10        | 15        |
| I, mg/kg      | 55        | 4.5       | 20        |
| Vit A, IE/kg  | 300000    | 300000    | 300000    |
| D, IE/kg      | 30000     | 15000     | 30000     |
| E, IE/kg      | 5000      | 8500      | 3000      |
| B1, mg/kg     | 250       | 250       | 150       |
| B2, mg/kg     | 200       | 200       | 75        |
| B6, mg/kg     | 200       | 100       | 150       |
| B12, mg/kg    | 2.5       | 1.5       | 3         |
| Niacin, mg/kg | 500       | 200       | 300       |
| Folate, mg/kg | 200       | 100       | 75        |

Table 20. Horse feed supplements used in Germany, claimed to be designed to supply at least one macromineral. Data are expressed as fed, assuming 880g/kg DM; units are g/kg for Ca, P, Mg, Na, K, Cl; mg/kg for trace elements, B1, B6, B12, niacin, biotin and carotin; IU/kg for vitamins A, D and E (Coenen, personal communication).

| n=30    | Ca    | P     | Mg    | Na    | Cu    | Zn      | Se     | I       |
|---------|-------|-------|-------|-------|-------|---------|--------|---------|
| min     | 0     | 0     | 1     | 70    | 15    | 20      | 1.5    | 1       |
| max     | 280   | 40    | 200   | 385   | 1500  | 7500    | 20     | 200     |
| average | 61.3  | 17.3  | 52.1  | 112.5 | 506.7 | 3783    | 8.6    | 85.5    |
| n=30    | Vit.A | Vit.D | Vit.E | B1    | B6    | B12     | Niacin | Biotin  |
| min     | 0     | 0     | 1.5   | 0     | 0     | 0       | 280    | 0       |
| max     | 75    | 0.04  | 1500  | 1000  | 3600  | 250,000 | 3500   | 200,000 |
| average |       |       | 760.2 |       |       |         |        |         |

Table 21. Horse feed supplements used in Germany, claimed to be designed in particular to supply vitamins. Data are expressed as fed, assuming 880g/kg DM; units are g/kg for Ca, P, Mg, Na, K, Cl; mg/kg for trace elements, B1, B6, B12, niacin, biotin and carotin; IU/kg for vitamins A, D and E (Coenen, personal communication).

| n=36    | Ca     | P     | Mg    | Na    | Cu    | Zn     | Se     | I      |
|---------|--------|-------|-------|-------|-------|--------|--------|--------|
| min     | 2.4    | 1     | 0     | 2     | 25    | 45     | 0.2    | 0      |
| max     | 31     | 17    | 80    | 67    | 1000  | 10,000 | 1250   | 125    |
| average | 18.2   | 8.7   | 15.83 | 18.2  | 687.6 | 4749.8 | 53.9   |        |
| n=36    | Vit.A  | Vit.D | Vit.E | B1    | B6    | B12    | Niacin | Biotin |
| min     | 0.2    | 0.02  | 2     | 40    | 40    | 40     | 150    | 2      |
| max     | 50,000 | 200   | 500   | 2,000 | 1,000 | 6,000  | 24,000 | 2500   |
| average | 1952.5 | 27.6  | 35.6  | 404.9 | 358   | 1768.2 | 2965   | 752.7  |

Table 22. Horse feed supplements used in Germany, claimed to be designed in particular to supply trace elements. Data are expressed as fed, assuming 880g/kg DM; units are g/kg for Ca, P, Mg, Na, K, Cl; mg/kg for trace elements, B1, B6, B12, niacin, biotin and carotin; IU/kg for vitamins A, D and E (Coenen, personal communication).

|         | Ca      | P      | Ca/P   | Mg    | Na    | Cu    | Zn     | Se  | I    |
|---------|---------|--------|--------|-------|-------|-------|--------|-----|------|
| min     | 20      | 3      | 0.7    | 2.4   | 8.5   | 55    | 153    | 1.3 | 0.5  |
| max     | 200     | 220    | 50     | 120   | 150   | 2000  | 11000  | 30  | 130  |
| average | 127.3   | 35.4   | 5.9    | 23.4  | 46.5  | 435.4 | 2315.8 | 8.9 | 26.8 |
| n=49    | Vit.A   | Vit.D  | Vit.E  | B1    | B6    | B12   | Niacin |     |      |
| min     | 100     | 2      | 80     | 8.5   | 5.5   | 45    | 45     |     |      |
| max     | 1200000 | 100000 | 7000   | 500   | 500   | 1250  | 2000   |     |      |
| average | 372827  | 36455  | 2785.5 | 134.1 | 123.1 | 620.9 | 588.8  |     |      |

## Feeding practices for the growing horses

Feeding practices can vary from country to country, but the basis for the growing horse is a ration composed of hay (very often first cut meadow hay), a mixture of simple feeds and a premix (or hay and a compound feed containing a mineral and vitamin premix). Obviously, the mineral and vitamin supply is depending on the nutrient levels of both feedstuffs and premixes. The above tables are referred to common feeding practices for growing horses in UK and Germany.

*Table 23. Feeding practices in U.K. for growing Thoroughbred and saddle horses (from Harris, 1999).*

| Type of Animal                    | % of Body weight on an as fed basis<br><i>Figures in italic refer to ratios of feed</i> |                                        |                  |
|-----------------------------------|-----------------------------------------------------------------------------------------|----------------------------------------|------------------|
|                                   | Forage (hay)                                                                            | Concentrates                           | Total            |
| Weanling foal 6 months : TB       | 0.75<br><i>40</i>                                                                       | 1.75<br><i>60</i>                      | 2.5              |
| Weanling foal 6 months : Non-TB   | 1<br><i>50</i>                                                                          | 1.5<br><i>50</i>                       | 2-2.5            |
| Yearling foal 12 months : TB      | 1.2<br>1-1.2<br><i>50</i>                                                               | 0.8<br>0.8-1.0<br><i>50</i>            | 2<br>2           |
| Yearling foal 12 months : non -TB | 1.2<br>1-1.2<br>1.25-1.5<br><i>70</i>                                                   | 0.8<br>0.8-1.0<br>0.5-1.0<br><i>30</i> | 2<br>2<br>2-2.25 |
| Long Yearling:<br>TB              | 1.2<br>1.25 –1.5<br><i>50</i>                                                           | 0.8<br>1-1.25<br><i>50</i>             | 2<br>2-2.5       |
| Long Yearling:<br>Non- TB         | 1.2<br>1.4<br><i>70</i>                                                                 | 0.8<br>0.6<br><i>30</i>                | 2<br>2           |
| Two year old : TB                 | 1.4<br>1.4<br>1.0-2.0<br><i>40 Racing</i>                                               | 0.6<br>0.6<br>0.5-1.0<br><i>60</i>     | 2<br>2<br>2-2.5  |
| Two year old : non TB             | 1.4<br>1.5<br>1.25-1.75<br><i>80</i>                                                    | 0.6<br>0.5<br>0.5-0.75<br><i>20</i>    | 2<br>2<br>2-2.25 |

Some practical examples of traditional rations for young horses are listed below from Miraglia (personal communication); some of them are not correct, especially in relation to the Ca/P ratio but are employed in practice for growing horse feeding.

In general, the classical rations used for young horses supply 1/3 to 2/3 of the calcium requirements and 2/3 of phosphorus requirements. The ration has to be supplemented at least to meet the minimum required. Little excesses of Ca can be accepted if the Ca/P ratio is correct and, at the same time, the basic levels of all the other nutrients are satisfied.

*Table 24. Feeding practice in Germany for growing Thoroughbred and saddle horses (Harris, 1999).*

| Type of Animal                              | % of Body weight on an as fed basis |              |           |
|---------------------------------------------|-------------------------------------|--------------|-----------|
|                                             | Forage (hay)                        | Concentrates | Total     |
| Weanling foal 6 months : Non-TB             | 1                                   | 1.0 – 2.0    | 2.0 – 3.0 |
| Yearling foal 12 months : non -TB           | 1-1.5                               | 1.0          | 2-2.5     |
| Long Yearling: Non- TB                      | 1-1.5                               | 0.5 - 0.8    | 1.5-2.3   |
| Two year old : non TB<br>depends if in work | 1.5 –2.0                            | 0.5-1.0      | 2 –3.0*   |

The mineral supplementation must be studied also in relation to the digestibility of the whole ration, the salts used and the peculiarities of the basal feedstuffs.

Examples of Ca and P supply for young horses in Italy (Miraglia, personal communication):

#### **Foal at weaning, live weight 225 kg**

The foal very often starts to eat not negligible quantities of solid feeds from the third month of age; nevertheless, it does not receive particular supplementations as it should have. At 6 months of age the milk assumption can be considered as less than 5 litres/day, while the ingestion of solid feeds varies between 2.5 and 3.0 kg/100 kg of body weight. The requirements can be considered 36 g Ca and 20 g P/day.

First example: milk + grass hay = 5 l of milk + 6 kg of hay

- Ca supply:  $(0.9\text{g/l} \times 5) + (3\text{g/kg} \times 6) = 22.5\text{g}$

- P supply:  $(0.6\text{g/l} \times 5) + (1.5\text{g/kg} \times 6) = 12\text{g}$

This ration is lacking about 14g of Ca and 8 g of P.

Second example: milk + alfalfa hay = 5 l of milk + 6 kg of hay

- Ca supply:  $(0.9\text{g/l} \times 5) + (15\text{g/kg} \times 6) = 94.5\text{g}$

- P supply:  $(0.6\text{g/l} \times 5) + (2.5\text{g/kg} \times 6) = 18.0\text{g}$

The calcium supply is in excess, while the phosphorus is still lacking.

Third example: post-weaning ration

crushed oats (3 kg) + grass hay of good quality (2.5 kg) + alfalfa hay (2.5 kg)

- Ca supply:  $(1\text{g/kg} \times 3) + (3\text{g/kg} \times 2.5) + (15\text{g/kg} \times 2.5) = 48\text{g}$

- P supply:  $(3.5\text{g/kg} \times 3) + (2.2\text{g/kg} \times 2.5) + (2.5\text{g/kg} \times 2.5) = 22.3\text{g}$

The calcium supply is more than adequate (little excess), phosphorus supply is adequate. The Ca/P ratio is however 2.15 (it should be 1.8).

These examples indicate that the more traditional rations used in Italy don't meet the Ca and P requirements of the foal; consequently, there will be a strong evidence that one or both minerals must be added to the basal rations. An early mineral supplementation is then necessary at least from 3-4 months of age, because of the considerable reduction of minerals in the mare's milk. Let now consider the yearling rations:

### **Yearling, average live weight 325 kg**

In this case the traditional rations supply about 2/3 of the Ca and P requirements; the supplementation will then cover the 1/3 lacking. In this period the requirements are 39 g of Ca and 22 g P/day.

First example: crushed oats, 6 kg + grass hay, medium quality 5 kg

- Ca supply:  $(1\text{g/kg} \times 6) + (3\text{g/kg} \times 5) = 21\text{g}$

- P supply:  $(3.5\text{g/kg} \times 6) + (1.3 \times 5) = 27.5\text{g}$

This ration is frequently used but it is very dangerous because it combines a calcium lack (18 g) to a phosphorus excess, with a Ca/P ratio of 0.76.

Second example: crushed oats, 6 kg, + grass hay, good quality 2 kg, + alfalfa hay, 3 kg

- Ca supply:  $(1\text{g/kg} \times 6) + (3\text{g/kg} \times 2) + (15\text{g/kg} \times 3) = 57\text{g}$

- P supply:  $(3.5\text{g/kg} \times 6) + (1.3\text{g/kg} \times 2) + (2.5\text{g/kg} \times 3) = 31\text{g}$

In this case the mineral amount is more balanced if compared to the 1st example; nevertheless, there is both a Ca and P excesses, with a Ca/P ratio of 1.8.

Let now consider, as general examples, some rations for weaned horses, 8 months of age (Table 25), and growing horses aging 20 months (Table 26), composed of first cut hay and oats, the same hay and a balanced compound feed, or hay, oats and a premix.

The minerals and vitamins requirements are taken from Tables 1, 3 and 4; the feed intake and the forage to concentrate ratios from Table 1 and 23. The data on oats composition are mean values of Table 11; the considered premixes are Product 1 and Product 3 of Table 19, administered dose is supposed to be 100 g/day; the compound feed considered is the mean value from Table 14.

As to the 8 month old horse, unbalances are common. In particular, Ca is in excess for ration 3 and lacking for ration 1 (hay/oats); moreover, very low amounts of Vit. A are provided by the basal feeds, and the addition of the premix is of the utmost importance. On the other hand, very high levels of Vit. E are also provided by both premixes considered. The minimum requirements are satisfied by diets 2, 3 and 4 (then excluding the traditional ration 1, hay and oats) for Zn, Mn, niacin and vit. B6 but large excesses can rise.

As to the rations for the 20 months old horse, the Ca and P supply is quite adequate for all the considered rations, excluding the one based on compound feed added premix (ration 7). The ration based on the same feedstuffs induced Ca excesses also for the 8 months old foal. Copper is lacking, the same is for Zn and Fe. The requirements of vit. A are met only by using premixes, as it is for vit. E. Unbalances are still present.

Table 25. Mineral and vitamin supply of different feeding plans for a 8 months old growing horses of mature weight 600 kg. In the second row, requirements (derived from previous tables).

|            | Requirements | Ration 1 <sup>1</sup> | Ration 2 <sup>2</sup> | Ration 3 <sup>3</sup> | Ration 4 <sup>4</sup> |
|------------|--------------|-----------------------|-----------------------|-----------------------|-----------------------|
| Ca (g)     | 44           | 21.4                  | 32.3                  | 75.9                  | 38.3                  |
| P (g)      | 24           | 11.6                  | 18.5                  | 34.0                  | 15.0                  |
| Mg (g)     | 12           | 7.4                   | 10.3                  | 16.5                  | 13.3                  |
| Na (g)     | 13           | 0.1                   | 5.1                   | 12.3                  | 4.1                   |
| Fe (mg)    | 600          | 350.0                 | 840.0                 |                       | 385.0                 |
| Cu (mg)    | 180          | 34.0                  | 123.5                 | 157.4                 | 118.5                 |
| Zn (mg)    | 450          | 206.9                 | 603.9                 | 964.9                 | 403.9                 |
| Mn (mg)    | 300          | 728.7                 | 1123.7                | 553.7                 | 873.7                 |
| Co (mg)    | 0.9          | 0.4                   | 1.3                   |                       | 1.8                   |
| Se (mg)    | 1.2          | 0.7                   | 2.7                   | 2.5                   | 3.2                   |
| I (mg)     | 1.2          | 0.4                   | 5.9                   | 7.6                   | 2.4                   |
| A (IU)     | 25000        | 2100.0                | 32040.0               | 126.5                 | 32040.0               |
| D (IU)     | 3000         |                       |                       | 11.6                  |                       |
| E (IU)     | 50           | 35.0                  | 534.0                 | 2.1                   | 334.0                 |
| B1 (mg)    | 12           | 21.0                  | 45.4                  | 47.7                  | 35.4                  |
| B2 (mg)    | 20           | 5.3                   | 25.1                  |                       | 12.6                  |
| Nia (mg)   | 60           | 63.0                  | 111.2                 | 202.8                 | 91.2                  |
| Pant (mg)  | 24           |                       |                       |                       |                       |
| B6 (mg)    | 6            | 5.6                   | 25.4                  | 43.8                  | 20.4                  |
| Chol (mg)  | 300          |                       |                       |                       |                       |
| Folic (mg) | 6            | 1.4                   | 21.4                  |                       | 8.9                   |
| B12 (mg)   | 60           |                       | 0.3                   | 210.8                 | 0.3                   |

<sup>1</sup>Ration composed of 3.5 kg DM first cut hay and 3.5 kg oats.

<sup>2</sup>Ration composed of 3.5 kg DM first cut hay, 3.4 kg oats and 0.1 kg of a premix (Product 1 of Table 19).

<sup>3</sup>Ration composed of 3.5 kg DM first cut hay and 3.5 kg of a compound feed added premixes (from Table 14).

<sup>4</sup>Ration composed of 3.5 kg DM first cut hay, 3.4 kg oats and 0.1 kg of a premix (Product 3 of Table 19).

## Conclusions

There is a huge variety of situations in the mineral and vitamin rationing feeding practices for growing horses in Europe. Some unbalances on Ca and P supply are still common in practice, mostly for the sporadic and incorrect use of compound feeds and premixes. It is also important to have good data about the composition of the feeds used and to use premix added compound feeds, or to add premixes. In this case, however, unbalances can rise, and excesses can also be a problem. The “perfect” product is not on the market, and the only choice for a balanced ration is to mix different feeds and supplements in adequate amounts, and to control the effect of the feeding plan in the field.

Table 26. Mineral and vitamin supply of different feeding plans for a 20 months old growing horses of mature weight 600 kg. In the second row, requirements (derived from previous tables).

|            | Requirements | Ration 5 <sup>1</sup> | Ration 6 <sup>2</sup> | Ration 7 <sup>3</sup> | Ration 8 <sup>4</sup> | Ration 9 <sup>5</sup> |
|------------|--------------|-----------------------|-----------------------|-----------------------|-----------------------|-----------------------|
| Ca (g)     | 42           | 48.2                  | 36.4                  | 98.7                  | 47.4                  | 53.4                  |
| P (g)      | 24           | 24.6                  | 19.2                  | 44.8                  | 26.2                  | 22.7                  |
| Mg (g)     | 10           | 16.2                  | 12.4                  | 22.9                  | 15.4                  | 18.4                  |
| Na (g)     | 15           | 0.1                   | 0.1                   | 14.1                  | 5.1                   | 4.1                   |
| Fe (mg)    | 1000         | 200.0                 | 400.0                 |                       | 890.0                 | 435.0                 |
| Cu (mg)    | 300          | 50.6                  | 49.2                  | 190.3                 | 138.8                 | 133.8                 |
| Zn (mg)    | 750          | 292.8                 | 294.6                 | 1161.0                | 691.6                 | 491.6                 |
| Mn (mg)    | 500          | 1365.6                | 1149.2                | 949.2                 | 1544.2                | 1294.2                |
| Co (mg)    | 1.5          | 0.2                   | 0.4                   |                       | 1.4                   | 1.9                   |
| Se (mg)    | 2            | 0.4                   | 0.8                   | 2.9                   | 2.8                   | 3.3                   |
| I (mg)     | 2            | 0.2                   | 0.5                   | 8.6                   | 6.0                   | 2.5                   |
| A (IU)     | 35000        | 1200.0                | 2400.0                | 144.5                 | 32340.0               | 32340.0               |
| D (IU)     | 6000         |                       |                       | 13.2                  |                       |                       |
| E (IU)     | 100          | 20.0                  | 40.0                  | 2.4                   | 539.0                 | 339.0                 |
| B1 (mg)    | 24           | 12.0                  | 24.0                  | 54.5                  | 48.4                  | 38.4                  |
| B2 (mg)    | 40           | 3.0                   | 6.0                   |                       | 25.9                  | 13.4                  |
| Nia (mg)   | 120          | 36.0                  | 72.0                  | 231.8                 | 120.2                 | 100.2                 |
| Pant (mg)  | 48           |                       |                       |                       |                       |                       |
| B6 (mg)    | 12           | 3.2                   | 6.4                   | 50.0                  | 26.2                  | 21.2                  |
| Chol (mg)  | 600          |                       |                       |                       |                       |                       |
| Folic (mg) | 12           | 0.8                   | 1.6                   |                       | 21.6                  | 9.1                   |
| B12 (mg)   | 120          |                       |                       | 240.9                 | 0.3                   | 0.3                   |

<sup>1</sup>Ration composed of 8 kg DM first cut hay and 2 kg oats.

<sup>2</sup>Ration composed of 6 kg DM first cut hay and 4 kg oats.

<sup>3</sup>Ration composed of 6 kg DM first cut hay and 4 kg of a compound feed added premixes (from Table 14).

<sup>4</sup>Ration composed of 6 kg DM first cut hay, 3.9 kg oats and 0.1 kg of a premix (Product 1 of Table 19).

<sup>5</sup>Ration composed of 6 kg DM first cut hay, 3.9 kg oats and 0.1 kg of a premix (Product 3 of Table 19).

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# **Influence of management and nutrition on growth in the young horse**

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## **Abstract**

Growing period in horses lasts between 3 and 5 years, depending on the genetic type and utilisation, that is to say 40 to 75% of the productive life of the horse. Athlete horses include horses destined to races and saddle horses destined to high level equestrian competitions. Race horses are expected to grow at a faster rate to reach mature weight at a much earlier age. In this case management, feeding practices and grazing management should be adjusted to support a high body development as permitted by their genetic potential; but such a growth often involves development diseases. Growth and bone characteristics are strictly linked to the evolution of live weight and are influenced by age, exercise and by the corresponding feeding strategies. Saddle horses destined to equestrian competitions are late-maturing animals; as a result growth management and the feeding practices include more or less compensatory growth. In the case of saddle horses destined to low level equestrian competitions and/or to leisure management practices, feeding systems and technical assistance are extensive as far as these classes of horses contribute widely to the exploitation of marginal lands in the extensive management practices.

*Keywords: growing horses, management, feeding practices, grazing*

## **Introduction**

A good feeding planning means the difference between developing a champion or just another horse. Young sportive horses are expected to become a top quality race and performance athletes. Management practices and nutrition planning of growing horses depend on the typology of horses and on the expectation of the breeders. Horses' husbandry and feeding systems are strongly determined by the goal of production and utilisation. Growing period in horses lasts between 3 and 5 years, depending on the genetic type and utilisation, that is to say 40 to 75% of the productive life of the horse. This situation involves a considerable economic and financial investment that influences performances, longevity and profits. Athlete horses include two big groups of horses: precocious horses destined to races and late-maturing saddle horses destined to high level equestrian competitions for which breeders support considerable investments (high quality mares and stallion, high level technical assistance, etc.). On the other side there are still late-maturing saddle horses destined to low level equestrian competitions and/or to leisure horses for which management practices, feeding systems and technical assistance are very different if compared to athlete horses because of their low economic value. These classes of horses contribute widely to the exploitation of marginal lands in the extensive management practices. Horses destined to sport activities (races or equestrian competitions) are bred with very intensive breeding systems. Race horses used to begin training at 15 months of age for racing as early as 24 months old while in saddle horses used to begin ridding at 3-4 years of age. In both cases training starts when growth and bone development are not completed. Foals of race breeds weight between 220 kg BW. at

weaning and 420-450 kg BW. at 18 months; sport and leisure horses weight between 225 and 275 kg BW. at weaning and 500-600 kg BW. at 5 years, depending on the genetic type. From birth to one year young horses reach between 55- 60% of the adult live weight and 70% of the adult size. Bone development reaches 80% of the total development at 18 months: this particular situation affects considerably osteoarticular pathologies, generally depending on nutrition imbalances and mistakes in feeding planning. The etiology of Developmental Orthopaedic Disease (DOD) is of multifactorial origins (McIlwraight, 2001), but nutrition plays the most important role in its development (Coger *et al.*, 1987); inadequate levels of proteins, minerals and energy could influence skeletal misdevelopment in young horses (Potter, 1982; Gibbs *et al.*, 1987; Savage *et al.*, 1993). Nevertheless, while the researches carried out in the last 15 years evidenced the importance of balanced rations in growing diets, most part of the horsemen continue to feed traditional rations generally composed by oats and hay only (Jones, 1989). Many nutrition-related disorders are usually a consequence of mismanagement systems, which work against rather than with nature, such as high carbohydrate, low fibre diet, sudden changes in feeding, etc. (McCarthy, 1990). But other aspects, together with the nutritional ones can expose horses, directly or indirectly, to growing problems: selection, feeding technologies, care and management.

## **Race horses**

Thoroughbred and Standardbred are expected meet a high body weight as early as possible. But those horses begins training and racing at 15 months and 24 months respectively. The consequence of this situation is that management and feeding practices, as also the nutrition planning, should be adjusted to support a high body development as permitted by their genetic potential. In fact, growth and bone characteristics are strictly linked to the evolution of live weight and are influenced by age, exercise and, consequently, by the corresponding feeding strategies.

## **Management and feeding practices**

The weight of young race horses ranges between 220 kg BW at weaning and 420-450 kg BW at 18 months (Willoughby, 1975; Martin-Rosset, 1983; Martin-Rosset, 1990; Pagan, 1998; Paragon *et al.*, 2000). In some specialized countries where thoroughbred breeding is much concerned, the foal would have been weaned as early as possible to allow its preparation for the foal sales. From the time the foal arrive at the new location, the purchaser will be concentrating his efforts in producing the horse for a yearling sale the following autumn: this will be a second significant deadline in the animal's life. Preparing a yearling for sale involves a careful monitoring of the horse's physique, reversing the trend of nature if it should affect the horse's limb conformation, and ensuring above all that the individual in offer meets with the approval of the buyers and their agents at the sale. Sale preparation is generally done over a two month period prior to the sales. The yearling are brought into stables, reintroduced to regular handling, routinely exercised to improve their condition – their programme flexible in accordance with their progress. After sales the yearling is transported again to a training or pre-training centre to be broken and then intensively prepared for a racing career. In this kind of breeding it is essential to understand that the breeding of race horses provides different challenges and targets to different breeders involving different management practices (Nugent, 1996). If foals are well managed before weaning, this phase will be not very stressful for them. In race horses, growth rhythms are very fast: foals could growth till 1600 g/day in the first month with an average value of 750 g/day till 6 months: horses reach, at 6 months, 50% of the adult weight. At 12 and 18 months of age race horses reach

respectively 66p100 and 80p100 of the final weight. If we consider that the birth weight is 10p100 of the adult weight, it is easily comprehensible the onset of growth pathologies if the nutrition planning and the managements and care practices are lacking. Horses are expected to grow at a faster rate now: in feeding practices this means that yearling race horses are fed high feeding plans as early as they are still suckling the mare; as a result foals and yearlings are offered high feeding level, probably 15 to 20 % over the requirements designed by INRA (Martin-Rosset, 1990); generally the ration of these horses is constituted by high quality forages (green forages OMD = 60-70%; hays OMD = 55-60%), supplemented with high percentages of concentrates: 40 to 60% in DM, depending on the forage quality, on the age and on the physical activity of yearlings. A good body development of young horses begins in the uterus of the mare: consequently, a good feeding planning of the mare is important to ensure a good weight of the foal at birth and a reduction of metabolic bone diseases. In these kind of horses a particular role is played by the administration of specific pre-weaning feeds that will favourite the passage from milk diet to the solid one. The young foal begins to taste the concentrate feed destined to the mare since the first days of life; nevertheless, the ingestion of this kind of feed, formulated to satisfy the nutrient requirements of the mare, doesn't satisfy those of the young foal: the consequence is an unbalanced ration for it. This problem could be limited using specific feeds and the administration could be easily practiced through the creep feeding system that provides an area, in outdoor conditions, and an appropriate feeder, in indoor conditions, where the foal can eat without interference from the mare. The concentrate destined to foals and administered with this technique has to be very palatable and it is necessary to eliminate the remainders from a day to another. The creep feed is very important because it influences the weaning date that, in these horses, tends to be precocious, from 4 to 5 months, and is largely influenced by the mare milk production both in terms of amounts and composition: a mistaken weaning has negative effects on the bone development with a reduction of cannon diameter and density (Warren *et al.*, 1998). Foals are supplemented between 3 months and weaning using the creep feeding technique to balance the low milk production (Doreau *et al.*, 1986) of mares and to adjust minerals, vitamins and amino acids allowances. The creep ration could be provided to the foal also earlier, mainly in the case of a low milk production of the mare; at the beginning the foal will not eat significant amounts of feed, but it gradually increases its intake. Weaning is one of the most important phases of the foal life. Immediately after weaning and between weaning to 12 months a good feeding planning needs of special supplements including minerals, vitamins and amino acids. In this period the nutrient requirements are very high and consequently horses are offered high feeding levels; nevertheless, a high feeding level, if not well monitored, could determine negative effects both in mares and foals; in the case of foals they are mainly constituted by bone diseases. The possibility to have such problems need a particular attention in the choice of feeds in relation to their characteristics and in the nutrition planning. Every stressful condition that alter the theoretical growth curve affect negatively the complete development of the horses and the expression of the genetic potential. Many researches carried out both in USA and Europe demonstrated that in the last 20-30 years the development of skeletal problems in young horses is more and more obvious (Knight *et al.*, 1985). This situation could come both from hypo or hyper nutrition that often origin the development of orthopaedic diseases (DOD) that include osteochondrosis, physitis or epiphysitis, flexural deformities (contracted tendons), etc.. Many other factors such as genetics, trauma, such as running on hard surfaces are likely to be involved in the development of the disease (Hoppe and Phillipson, 1984; Wagner *et al.*, 1985). But nutrition plays the most important role in developing them. Overfeeding is frequently blamed in development of these kind of diseases, mainly in foals that are growing rapidly. The overfeeding comes from the need to breed horses more and more precocious: breeders try to reach the maximum development in the

minimum time: the consequence generally originates overfeeding and nutrient imbalances. In the case of race horses, indeed, it would be better to reach the optimal development and not the maximum development: the maximum development corresponds to that of horses destined to meat production (Wolter, 1994). The negative effects of overfeeding is amplified if feeds used in the diet are characterized by minerals imbalances, lacks in essential amino acids, excess of carbohydrates etc. (Stromberg, 1979). Excessive energy intake or calcium intake should be avoided but the diet should be evaluated and balanced for calcium, phosphorus, copper and zinc (Hurtig *et al.*, 1993). In this context, a controlled nutrition planning concerning energy and proteins allowances near to the requirements but absolutely not over them, in which the requirements are met using dietetic feeds and high quality supplements, could be suggested. An experimental controlled and balanced nutrition planning in thoroughbred from weaning till 18 months was carried out in the same stud farm over 12 years (Miraglia *et al.*, 1996; Polidori *et al.*, 2000); this nutritional planning permitted to reach at 18 months of age the standard values of weight without affect negatively both development and the future performances. 12 years ago, at the beginning of these experiment many negative aspects concerning management and nutrition were observed. In particular, there was not a schedule planning in all the management practices of the stud farm: nutrition was only a marginal aspect and the high percentage of bone diseases were considered almost a normal phenomenon and the veterinary came in the stud only when pathologies were evident. This situation is still frequent now in a high percentage of race horses and saddle horses farms. The changes that were progressively carried out in the stud were the following:

- use of specialised horse technicians to control the feeding planning and the animal welfare;
- monthly checks of horses for:
  - \* general welfare conditions;
  - \* live weight;
  - \* clinical conditions, including radiographic controls in correspondence of the critical development period (6-12-15 months of age);
  - \* parasites controls;
  - \* feet conditions;
- ration formulation in relation to monthly controls, sometimes head/head: in fact, in the case of excessive or low live weight and increase of bone diseases, horses were kept out by the herd to allow an individual ration with adequate dietetic feeds and supplement useful for the specific problem;
- improve in breeding planning: selected mares and stallions were progressively introduced by the breeder.

During this experiment a critical period of growth occurred when horses were 12 months old; the balanced ration permitted to have a compensatory growth in the following months without bone diseases. The success of this experiments permitted to the breeder to improve considerably the quality of his horses as demonstrated by the evident decrease in diseases development, as shown in Table 1, and in the increase of breeder's prices during these years.

In the last years the thoroughbred coming from this farm became more and more clever and a lot of other breeders with a limited number of foals asked to breed their horses there till the sales. This kind of feeding agrees to manage the growth of the foals with particular nutritional strategies and has a fundamental role in the planning of winter feeding, a period that associates a low availability of forage together with physiological growth crises and consequent risk of bone injuries. Nevertheless, if the restricted feeding planning is not well monitored as above mentioned, it is possible to have development diseases (Ellis and Lawrence, 1978). In this case horses will not reach the standard adult weight and the tissues

development will be longer and horses will not be worked as hard or as soon as properly fed horses (Knight *et al.*, 1985).

*Table 1. Bone diseases, underweighted horses and race performances at the beginning of the trial and after 6 years (Miraglia *et al.*, 1996).*

| Years                                             | 1989 | 1994 |
|---------------------------------------------------|------|------|
| N. of heads                                       | 14   | 24   |
| Underweighted foals<br>(6-7 months of age) (%)    | 100  | 25   |
| Bone diseases at weaning (%)                      | 78.6 | 25   |
| Underweighted yearlings<br>(18 months of age) (%) | 28.6 | 22.7 |
| Bone diseases at 18 months (%)                    | 21.4 | 4.5  |
| Performances in races                             |      |      |
| - never in race (%)                               | 35.7 | 9.7  |
| - never placed (%)                                | 42.9 | 22.6 |
| - winnings and/or placed (%)                      | 21.4 | 67.7 |

### **Management and feeding practices: the role of pastures in race horses breeding**

Grazing and exercising on pasture are of much concern namely for foals and yearlings to stimulate bone and muscle development. But quality of pastures is quite contrasted in Europe due to geographical area. Consequently the exploitation of pastures in race horse breeding is different; good pastures can play an important part of the horses care program. Pastures can be used as exercise and exhibition paddocks playing no role in nutrition planning; in other cases, high quality pastures have an important role in nutrition planning during most part of the year. These pastures offer several advantages because the forage has an high digestibility, requires no storage, limits labour and provides exercise. These different situations depends on the breeders choice but are largely influenced by the geographical area. In fact, in central and northern European countries the considerable grazing surfaces, generally characterized by high quality forages, influences the nutrition planning; in other cases, namely in southern European countries, the pastures characteristics are affected negatively by climate and environmental factors with a considerable incidence of summer dryness; in this case the nutritive role of pastures is limited and it is fundamental only in exercising. In fact exercising influences the muscular growth and, consequently, good athletic performances at the training beginning: this means that horses destined to competitions need of high surfaces, also if the quality of pastures is low: consequently, it is important to have low value of stocking rate. In spite of the fact that race horses breeding is based on an intensive pasture system, with a socking rate of about 1.5-2.0 horses/ha, in practical feeding conditions, and specially in northern-central European countries where race horses breeding is much concerned, the stocking rate is lower, generally 0.7head/ha. In practical field conditions, the breeders that dispose of high quality pastures and high surfaces, consider their pastures as a "panacea" and often foals are fed only with pastures till weaning and even till 12 months of age, without any supplementation in minerals or other supplements. In this case it is possible to have an increase in bone diseases, depending on the mineral losses determined by diarrhoea phenomenon caused by the ingestion of large amounts of green grasses (mainly in spring time) without supplements addition; this situation can become worst if the *gramineae*

constituting the pasture are high in oxalates and in acid phytic: in this case it should be better to maintain high the Ca:P relationship. For this reason an important role in the nutrition of young horses, specially of those having very precocious development and for which pastures play an important role in the total ration, is played by supplements and dietetic feeds that are more and more employed in the feeding of high quality horses. In the case of race horses breeding it is necessary to by-pass an old fashioned concept based on the traditional feeding plan where the diet is constituted by hay, oat and pasture. The possibility to evaluate in a correct way the diet planning introducing high quality feeds supplemented in balanced allowances of minerals, vitamins and essential amino acids agree to reach high performances horses. Many important management practices involve the pasture care and the production of high quality green forage; for instance, the soil should be tested to determine the plant nutrient needs for optimal production. Also, a proper fertilisation will improve protein content, increasing tolerance to dry weather, re-growth after grazing and resistance to disease. After the pasture is established and is being grazed, several important management practices should be followed; periodic scattering and/or removing of dung piles will help to reduce the parasite population. In fact it is important to remember that pasture represents the intermediate phase of the parasites cycle and that larvae are ingested with grass. Obviously, the parasite problem will be reduced if all horses are de-wormed before they are introduced in the pasture and following a regular plan of treatments. A new pasture should not be grazed until roots are well established and at no time should the pasture be overgrazed to the point where not enough leaf material is left to permit a correct re-growth (Cottarelli, 1989). In race horses breeding pasture have to be grazed on a rotational basis and during the rest period it will be possible to do the care practices of the sward (Miraglia *et al.*, 2000). The time of re-growth in an intensive pasture system depends on the season period: in fact, this time will be of 20-25 days between April and June, increasing to 30-40 days in the following months (Trillaud-Geyl *et al.*, 1990) and it is largely influenced by the summer dryness that is more or less evident depending on the different European countries. Another important aspect in management pastures destined to race horses come from the fact that horses tend to overgraze portions of a pasture and undergraze others. Alternate grazing with cattle or small ruminants will determine a better exploitation of pastures; in fact cattle and horses tend to avoid grazing grass that grows around their own droppings but will graze around each other's dropping (Miraglia *et al.*, 2000). Another problem that can arise in management practices derives from the hierarchy establishment between horses: it is important to be sure that an adequate pasture area is available to prevent aggression phenomenon. Nevertheless, in race horses breeding, when horses reach 12 months of age it is necessary to separate males from females; in the constitution of groups it would be better to not exceed 6-7 heads, also if surfaces is abundant because aggression phenomenon by dominants horses are very frequent and often determine excessive concentrate consumption of the dominant and less consumption of timid horses as well with consequent cases of bone diseases in the former (Garbers and Blanco, 1987). The constitution of groups should takes into account a deep knowledge of the characters of the young horses: this will agree to constitute groups in which aggressive horses are separated by less aggressive horses, mainly if the concentrate administration is practiced outdoor. In this case the concentrate amount has to be increased of 10-15% because it is necessary to consider a waste quota. The constitution of groups is a very delicate phase: in fact breeder has to preview the constitution of groups with particular problems like development bone diseases or growth crises that need of particular care and a specific nutrition planning. Another aspect to take into account in group constitution (sex, character) is the age; generally a good system could be to constitute groups having almost the same age, generally to put together the yearlings born between January and March and another constituted by yearlings born between April and May/June. On the other side, in the case of horses with development diseases

having the necessity to limit or to increase the amount of concentrates, it would be better to have some group constituted by horses with different ages but that need of the same ration; e.g. fat horses or horses with bone diseases need to have a restricted amount of concentrates (mainly if the diet is constituted in large part by cereals) can be introduced in the group of younger horses having lower requirements in term of amounts. In alternative, horses with developmental orthopedic diseases, both fat and thin horses, can be isolated from the group for limited period using little paddock where the young horse can exercise. Another aspect that can lead to some doubt in the care of young race horses concerns the time spent at pasture; two typologies can be practiced: horses can be kept at pasture only during the day or 24h per day. In the first case it is possible to have a strictly control of the horses and the administration of concentrates is done in box before coming out at morning and in the evening when they come back: in this way an individual feeding can be provided, if necessary; in the other case horses can be kept outdoor day and night but it is important to dispose of outdoor boxes, adequate grooming assistance and of particular feeders for the administration of concentrates and of supplements. In this case individual feeding can be provided if the stud farm dispose of sufficient grazing area to constitute many groups of yearlings having the same requirements and that need of the same supplementation with dietetic feeds and/or supplements. Any case, in these conditions it is necessary to put indoor the horses once a month for the routine practices: live weight control, veterinarian controls, parasites controls, feet care, adjustment of the diet, etc. Both these two typologies have advantages and disadvantages on growth: in the case of the use of pastures only during the day it is reduced the time spent in exercising and this will reduce the muscular development; on the other side, in this case it will be possible to control strictly the administration of feeds; in the other case horses will benefit of a considerable exercise improving the development; nevertheless, if management practices will be lacking, the feeding planning, the care and welfare conditions could be compromised.

Finally, the most important management and feeding practices that influences the development of the young horse destined to races with very precocious development are:

- high technical assistance (grooming, nutritionists, veterinarians, etc.);
- good feeding planning of the mare;
- appropriate evaluation of the quality and of the amount of milk produced;
- appropriate evaluation of the nutritive value of green (pastures) and harvested forages;
- monthly control of weight starting from that of birth that agree to quantify the daily BW increase, the difference with the theoretical values and, consequently, the adjustment of the feeding planning and eventual use of specific supplements;
- use of creep feeding technique to give to the foal a balanced special pre-weaning feed;
- feed in individual feeder whenever possible;
- feed balanced rations according to requirements and include either hay or pastures;
- use appropriate care systems in pasture management (rotations, control of infestant species, safeguard from parasites diffusion in the sward, fertilisation, etc.);
- reduce (limit) the use of single cereals, namely oat, that predispose to unbalanced growth and onset of bone diseases;
- verify excesses and lacks of each nutritive element and the ratio between them;
- verify monthly the bone development through clinic controls (using, in some case rx controls) and parasites incidence;
- keep feed rooms locked.

## Saddle horses with late maturing development

Saddle horses used for sport and leisure activities are fairly late-maturing animals if compared to race horses; consequently, in this case breeding practices are not influenced by the precocious birth date because these horses are used either at 4 or 6 year of age for those destined to sport activity: show jumping, three days events, dressage and endurance respectively. Leisure horses are provided either from a by-product of sport horses production or from a specialised production which is arising since this last decade in different European countries. Sport horses and leisure horses account for an important business. The late maturing development influences the nutrition planning and the choice of the feeding level both for mares and young horses referred to the feeding strategies useful to calculate appropriate amount and proportions of forages and concentrates to be used to meet the requirements.

### Management and feeding practices

The management of saddle horses follows two different kinds of productions: saddle horses destined to sport and saddle horses destined to leisure. The most important difference concerns the economic aspects: as for race horses, saddle horses destined to sport represent a considerable economic investments, starting from the use of high genetic levels of mares and on the use more and more diffused of the artificial insemination: the economic value of young horses increases progressively from conception to competitions: the fact that the life at the stud farm is longer than that of young race horses, involves a consequent increase of the risks of injuries, developmental orthopedic diseases, etc.; consequently, it is necessary that the breeder uses all the technical assistance available to limit such risks. In this typology of horses the management practices are very close to that used in race horse breeding. Nevertheless, the fact that these horses are late-maturing animals, imposes different choices for what concerns the growth management, the exploitation of pastures, the nutrition planning and the kind of diet. Mares destined to the production of sport horses are fed high quality forages (OMD=50-55%) to meet 100% of the requirements. During the growing period, feeding and management expenses can account for a large part of the production costs but to a lesser extent for the very high value of horses used for equestrian competitions. In consequence some experimental trials were carried out in USA and in France (NRC, 1989; INRA 1984-1990) to propose different feeding levels with different growth in relation to the production goal (sport vs leisure horses), to the economic value of horses and to the kind of utilisation (equestrian competitions vs leisure). In an attempt to reduce costs, forages and grazed herbage are used to manage longer growth curves using more or less compensatory growth (Figure 1). Long term experiments carried out in France concerning the management and feeding of French saddle horses over many years (Trillaud-Geyl *et al.*, 1986; Bigot *et al.*, 1987; Trillaud-Geyl *et al.*, 1990; Trillaud-Geyl *et al.*, 1992; Micol and Martin-Rosset, 1995) had the aim to investigate growth and development of late maturing saddle horses, fed different winter forages and different systems of grazing management during the grazing season using different compensatory growth. These studies demonstrated that young horses had a growth compensation during the summer when they had a limited level of feeding during the foregoing winter: growth compensation was as high as the level of feeding was low. But live body weight and development at 3.5 years of age were not significant between well fed and restricted horses. In this case grass provided 50 to 60% of total food supply of the young horse and 60% of total weight gain. In these trials horses were fed indoors during the winter with forage-concentrate diets; they grazed for 160-180 days on natural pasture sward in a temperate climate. Live weight gains in winter decrease with age (0.60, 0.31 and 0.19 kg/d at



1, 2 and 3 years old that means a decline of daily gain with age from 600g/d to 50g/d between the first and the third grazing season and it is influenced by the live weight gain obtained during the previous winter period. Radiographic controls carried out on these horses showed that young horses with high growth values during the second year of life were more affected by bone diseases than those having lower growth rhythms as previously demonstrated by Cymbaluk *et al.* (1990).

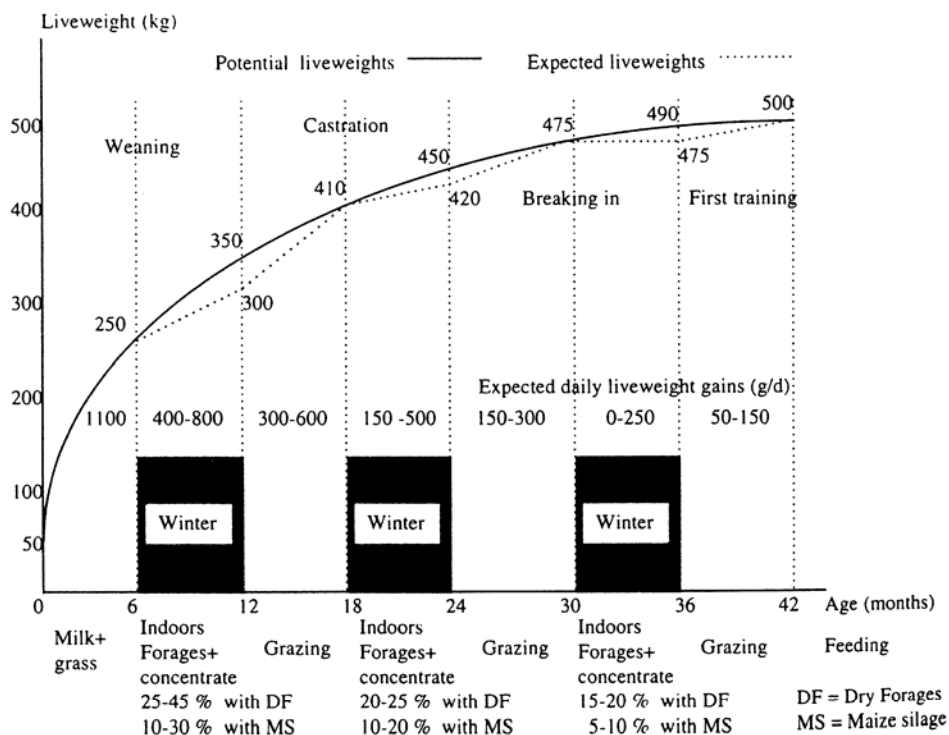


Figure 2. Feeding management of growing saddles horses (According to Bigot *et al.*, 1987; Trillaud-Geyl *et al.*, 1990; INRA, 1990).

In the case of saddle horses destined to leisure, management and feeding practices are very different and are mainly linked to the exploitation of extensive surfaces in humid temperate mountainous or hilly areas and/or rangelands where mares and young horses are bred outdoor during the grazing season. In both these situations grass and preserved forages are extensively offered to the mares in association or not with ruminants (Institut du cheval, 1987). Grazed feed resources have to meet 80% of the total requirements over the 7 months lactation period: the feeding level can be low (80% of the energy requirements) if body condition at foaling is good and if herbage availability is good. Growing horses are fed with medium quality hays (OMD=50%) supplemented with a limited proportion of concentrates (5 to 30% DM) (Micol and Martin-Rosset, 1995).

An extensive management system that can be adopted for mares of medium quality level: during winter, hays or maize silages (30-35% DM content) with low concentrate supplementation are offered outside: voluntary intake of the mares and growth rates of the foals depend on the quality of the hay fed to mares around foaling (Doreau *et al.*, 1990). The

grazing season lasts 230 days and no supplementation is given to the mares and to the foals during this period. The growth rates of foals are high at the beginning of this grazing period (1.55 kg/d) and decrease thereafter up to weaning in the autumn. In the case of saddle horses of low-medium quality, of those destined to leisure and of young fillies destined to reproduction, if the grazing surface available is high and the quality of pastures is good (case of French humid temperate regions), it is possible to use common grazing models both for saddle horses and for heavy horses (Martin-Rosset and Trillaud-Geyl, 1984): the animal response depends on the amount of grass available in spring and on the stocking rate practised. However, this production system is fragile and depends on good pasture resources and climatic conditions and needs of a concentrate supplementation of foal during the late grazing season.

### **Management and feeding practices: the role of pastures in saddle horses breeding**

Horse is a herbivore well adapted to make use of plant resources. Its ability to utilise large amounts of fibrous forages, often of low nutritive value, make it grazing and foraging in extensive feeding systems. Horses have several adaptive abilities to maintain and perform in harsh conditions when the total nutrient requirements can be achieved on a long term period. The ability of the horse to be managed in high forage feeding systems is explained in a major part by the distinctive features of the horse, which enables it to select, consume and digest forages and grazed resources. In saddle horse breeding the role of pasture is fundamental, mainly in the case of horses destined to leisure or to low level equestrian competitions. As already mentioned above, in the case of saddle horses destined to high level competitions the role of pastures is similar to that of horses destined to races; semi-extensive or intensive systems are used in pastures close to the stud farms to permit management practices and feeding planning useful for the reproduction performances (e.g., success in artificial insemination), for increasing milk production, for the correct development of the foetus in the last three months of gestation and to control the general welfare conditions. For the other typologies we refer to high forage systems where the pastures represent the most important source of feeding during the year. In these conditions the grass intake depends on its ingestibility, on the time of grazing, on the grazing activity, on the quality of plant resources, on the landscape use especially with other herbivorous species. Consequently, it is important to improve grazing performances and pasture management. In free-ranging conditions, horses spend 50-70% of their time to consume available food resources and only 20-30% for other activities. This ingestive activity is well distributed over the day during several grazing meals; important grazing period also occur during the night (Martin-Rosset *et al.*, 1978; Doreau *et al.*, 1980); grazing time can increase in autumn and in winter (Duncan, 1980) and the length of grazing is in relation to fibre content of the sward (Duncan, 1992). Extensive systems refer to the exploitation of natural pasture and uplands or of lowlands; in the first case pastures are characterized by low quality with poor productivity and where the total requirements can be met using low grazing intensity, with a stocking rate of 0.3-0.5-0.7 animal/ha, depending on the grass availability (Micol and Martin-Rosset, 1995; Miraglia *et al.*, 2001b). These pastures are characterized by many plant species sometimes with a high fibre percentage; gramineae and leguminosae plants are associated to many other kind of plants and to a shrubby vegetation (*nardus stricta*, *vaccinium myrtillus*, *carpinus spp.*, etc.) (Martin-Rosset *et al.*, 1981; Miraglia *et al.*, 2001). In these areas it is frequent the diffusion of infesting vegetation with different characteristics depending on the area (wet or arid areas) (*Ranunculus spp.*, *Taraxacum officinalis*, *Plantago spp.*, *Rumex*, *Juncus spp.*, etc.) (Miraglia and Bocchi, 1982; Catalano and Miraglia, 1985; Miraglia, 1989; Miraglia *et al.*, 2000). In the semi-extensive system of temperate climatic conditions, grazed herbage is a relatively cheap resource for

growing saddle horses. Young horses graze natural swards receiving a moderate nitrogen fertilisation (80-150 kgN/ha). Rotational grazing and cut of surplus herbage are involved. Stocking rate achieve 2.0-2.5 horses/grazed ha for young horses and decreases with age and weight of animals (Micol and Martin-Rosset, 1995). In synthesis the general management practices to observe in saddle horses breeding are:

- appropriate evaluation of the quality and of the amount of milk produced;
- appropriate evaluation of the nutritive value of green (pastures) and harvested forages;
- feed balanced rations according to requirements and include either hay or pastures and verify the possibility to realize different growing curves with different diets;
- use appropriate care systems in pasture management (rotations, control of infesting species, safeguard from parasites diffusion in the sward, fertilisation, etc.);
- use periodic control about the parasites incidence in the young horses.

## Conclusions

The most important management and feeding practices that influences the development of the young horse depend on the production goal and on the kind of activities; two big groups are identified: on one side we have race horses and saddle horses destined to equestrian competitions and on the other we have saddle horses destined to low levels equestrian competitions and to leisure. In the first case the difference between race and saddle horses is only in the growth precocity, but the management and the feeding practices are almost the same with the difference that young saddle horses remain at the stud for more time: high technical assistance, good feeding planning of mares and young horses according to requirements including also hay and pasture, appropriate care systems of pastures, monthly health controls and all the other factors previously mentioned are fundamental to produce high performance horses. In young horses destined to competitions it is important to underline the very early bone development during the first year that is strictly linked to the evolution of body live weight, to the age and to the exercise and, consequently, to the corresponding feeding strategies that determine the incidence of development diseases and performances. In this context an important role is played by the management practices in the most relevant growth crisis periods, namely when foals are aged 6 and 12 months. The use of special technologies, like creep feeding in the pre-weaning phase, dietetic feeds, appropriates supplements, etc., a correct exploitation of the pastures (kind of pasture, stocking rate, parasites controls, pasture care, etc.) and, finally, a continuous healthy monitoring of horses from specialised veterinarians will determine the difference in develop a champion or just another horse. Nevertheless, in spite of the fact that the scientific knowledge is quite advanced in identifying the best breeding technologies, in practical field conditions it is difficult to find a proper use of them. In the case of late-maturing horses destined to low level equestrian competitions and to leisure, growing horses can be fed different types of diet with an use more or less important of forages, mainly pastures, because of their considerable capacity to develop compensatory growth. Forage diets vary considerably in relation to the climatic conditions and to the available resources; the variations that can occur during the grazing period influence the nutrition planning and, consequently the feeding strategies that, in this case, correspond to different nutrition levels to reach more or less compensatory growth. So, it will be possible to have different kind of breeding, ranging from extensive systems in rangelands or harsh conditions to the semi-extensive systems. Nevertheless, a better knowledge about the long term effects of these systems on growth and on development diseases in young horses is needed. It will contribute to improve grazing, forages management and, consequently, an appropriate choice of the kind of diet namely the level of concentrate supplementation in relation to the growing performances.

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# Studies on the metabolic situation of mares and their foals during an early period after birth

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## Introduction and objectives

Birth itself and the early period after it is related to a striking change of the metabolic situation in the neonate and stress in the dam. In equines, clinical-chemical data about this period are rare. For that reason, this study was intended to investigate the metabolic situation of mares and their foals in an early period after birth.

## Material and methods

Fifteen trotter mares and their foals were used in this study. Blood was collected immediately after the first sucking (t1:  $2.37 \pm 1.36$  hrs *p.n.*) as well as 6 (t2) and 12 (t3) hrs *post partum* / *post natum*. Blood sera were analysed for variables of the metabolism of proteins (total protein, albumins,  $\alpha$ -,  $\beta$ - and  $\gamma$ -globulines, urea), carbohydrates (glucose, fructose, lactate), fats (free fatty acids, triglycerides [TG], phospholipids, cholesterol,  $\alpha$ -,  $\beta$ - and pre- $\beta$  portions of the lipoproteins [ $\alpha$ -,  $\beta$ - and pre- $\beta$ -LP%]), vitamins ( $\alpha$ -tocopherol [ $\alpha$ -T],  $\beta$ -carotene, total retinol [TR]) and minerals (calcium [Ca], magnesium, inorganic phosphorus [ $P_i$ ], sodium, potassium, chloride) including total bilirubin and selected enzymes (glutamic acid dehydrogenase,  $\gamma$ -glutamyl transferase, aspartate aminotransferase and creatin kinase [CK]).

## Results and Discussion

In the mares, it seems that most of the investigated parameters were not clearly effected by the birth. Also within 12 hrs *p.p.* there were predominantly no marked alterations. Lactate and CK decreased ( $P < 0.05$ ) and increased ( $P < 0.05$ ), but within the normal range.  $\beta_2$ -globulines were generally low, possibly why the mammary gland selectively concentrates proteins *ante partum*. The striking low concentration of Ca and  $P_i$  at t1 and their further drop ( $P < 0.05$ ) caused no clinical signs. Untypical for horses on pasture, like the mares in this study, was the low  $\beta$ -carotene concentration in the blood serum. Contrary, the concentrations of TR and  $\alpha$ -T were normal. In the foals, all investigated protein fractions, urea and glucose increased ( $P < 0.05$ ) after sucking while fructose decreased ( $P < 0.05$ ). TG and  $\beta$ -LP% at t1 were higher than in the mares ( $P < 0.05$ ) and additionally rose until t3 ( $P < 0.05$ ). In general, the blood lipids were more similar to LDL- than to adult HDL-type animals. TR and  $\alpha$ -T were much lower than in dams and only  $\alpha$ -T increased after sucking. The concentration of  $\beta$ -carotene was lower than detectable. Ca, but not  $P_a$ , was striking low at t1 and rose markedly until t3 ( $P < 0.05$ ). Bilirubin and all measured enzymes increased *p.n.*, but within the individual normal range.

## Conclusions

In mares, the concentrations of Ca and Pi in the blood serum remarkably decrease after birth, but without any clinical reaction. Further investigations should consider the ionised part of Ca and the endocrine situation. In foals, the change from LDL- to HDL-type does not occur within 12 hrs *p.n.*. It is not exactly known when it happens and whether modifying factors act.



# Feeding practice in Hanoverian Warmblood mares and foals with regard to the incidence of osteochondrose

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*Keywords: horse, feeding, minerals, osteochondrosis*

## Introduction

Northern Germany is a traditional region for horse production focussed on the Hanoverian Warmblood (HW). To improve the quality of the breed, information regarding health and production management is necessary. The objective of the present study was to obtain valuable data on feeding systems, feed composition and nutrient intake in mares and foals and the corresponding incidence of osteochondrotic lesions. This study was part of a collaborative survey on the incidence of osteochondrosis in the HW.

## Material and methods

Totally 629 HW foals born between December 26, 2000 and July 01, 2001 (308 males, 321 females) and their dams from 83 farms in Germany were included in this study. Over a period of six month foals were weighed monthly on a portable electronic scale. Simultaneously feed samples were obtained and the nutrient intake of the rations was recorded during stable period and pasture time. Feed samples were analyzed for crude nutrients, macro and trace elements. For the mares, amount of feed and the analysed nutrients in the feed samples were used to estimate nutrient intake during stable period. During pasture time nutrient intake by grass regarding dry matter intake was calculated for the mares. As the foals were not fed separately from their dams during stable period and pasture time, the nutrient to energy ratio (stable ration for the mares, standard milk yield, and grass) was calculated to assess the supposed nutrient intake in foals. This proceeding implied an adequate energy intake by the foals regarding the individual growth rate. X-ray controls for osteochondrosis (OC) was done between the fifth and tenth month after birth. Radiographic diagnosis showed 226 foals with signs of osteochondrotic lesions.

## Results and discussion

During the stable period horses received rations based on roughage, concentrates and minerals (Table 1). In the mare the intake of digestible crude protein (DCP) and digestible energy (DE) during the first 2 month of lactation (stable period) was below the requirements (deviation >10 % of the recommendation, GEH 1994) for DCP in 70 % and for DE in 51 % of the mares. During pasture time a surplus in DCP and DE was calculated for the lactating mares. During stable period 26 - 40 % of the mares showed a mineral intake (Ca, P, Cu, Zn) which was

significant below the recommendations (deviation > 10 % of the GEH 1994). A similar result was obtained for pasture. During stable period there was no separate feeding of the foals, but in generally they participated in the meals for their dams. The foals were mostly on pasture from the third month of life and the precise nutrient intake was unknown. Milk and grass delivered the energy and nutrients, but nearly no other supplements were given to the foals. Considering the ratios of nutrients to energy, an insufficient Ca, P, Cu, and Zn supply was observed for nearly all foals. However, there was no link between feeding or mineral status and the incidence of osteochondrotic lesions in the foals. Foals with or without osteochondrotic lesions were monitored under the same feeding and management regime on the farms. The question if the incidence of osteochondrosis might be lowered by an optimized mineral intake remained open. It should be pointed out that the management of the mares and foals needs to be optimized. The protein supply by the typical hay and/or silage rations was clearly below the recommendations. On pasture foals and mares need an adequate mineral supplementation to optimize the mineral intake.

*Table 1. Crude nutrients in feedstuffs for lactating mares and foals.*

| N                | Grass<br>185 <sup>1)</sup> |     | Hay<br>76 |     | Silage<br>63 |      | Grain<br>14/72 <sup>2)</sup> |     | Concentrate<br>87 |      | Minerals<br>52 |      |
|------------------|----------------------------|-----|-----------|-----|--------------|------|------------------------------|-----|-------------------|------|----------------|------|
|                  | mean                       | SD  | mean      | SD  | mean         | SD   | mean                         | SD  | mean              | SD   | mean           | SD   |
| DM <sup>3</sup>  | 216                        | 49  | 868       | 24  | 636          | 142  | 874                          | 14  | 878               | 17   |                |      |
| ash <sup>4</sup> | 104                        | 28  | 78        | 23  | 92           | 35   | 29                           | 12  | 88                | 38   |                |      |
| protein          | 182                        | 49  | 111       | 39  | 141          | 41   | 128                          | 12  | 137               | 20   |                |      |
| fat              | 34                         | 9   | 18        | 6   | 25           | 8    | 36                           | 9   | 28                | 5    |                |      |
| fibre            | 219                        | 32  | 314       | 30  | 294          | 35   | 84                           | 12  | 112               | 58   |                |      |
| NfE              | 461                        | 53  | 479       | 43  | 448          | 54   | 724                          | 21  | 634               | 79   |                |      |
| DCP              | 128                        | 38  | 65        | 25  | 85           | 29   | 111                          | 11  | 113               | 17   |                |      |
| DE               | 11.9                       | 0.8 | 9.7       | 0.7 | 10.2         | 0.8  | 15.7                         | 0.4 | 14.1              | 1.3  |                |      |
| Ca <sup>5</sup>  | 3.8                        | 1.3 | 4.8       | 1.3 | 5.3          | 2.2  | 0.7                          | 0.5 | 19.1              | 13.1 | 136.6          | 49.0 |
| P                | 4.1                        | 0.7 | 3.0       | 0.8 | 3.7          | 0.9  | 3.9                          | 0.4 | 6.5               | 4.4  | 32.0           | 14.5 |
| Mg               | 2.0                        | 0.4 | 1.8       | 0.7 | 2.1          | 0.6  | 1.2                          | 0.1 | 3.0               | 1.6  | 19.9           | 9.9  |
| Na               | 1.1                        | 0.8 | 1.8       | 1.7 | 2.2          | 2.0  | 0.4                          | 0.4 | 4.9               | 3.4  | 52.6           | 20.2 |
| K                | 30.4                       | 8.3 | 19.2      | 8.2 | 26.2         | 10.7 | 5.0                          | 0.7 | 13.1              | 4.8  | 5.2            | 3.1  |
| Cl               | 9.9                        | 4.7 | 8.6       | 4.0 | 10.0         | 5.7  | 1.1                          | 1.1 | 10.1              | 9.8  | 68.5           | 38.5 |
| Cu <sup>6</sup>  | 10                         | 3   | 7         | 3   | 8            | 3    | 5                            | 3   | 50                | 53   | 698            | 352  |
| Zn               | 37                         | 11  | 27        | 10  | 37           | 18   | 31                           | 12  | 238               | 242  | 3680           | 1899 |
| Fe               | 376                        | 495 | 374       | 783 | 350          | 402  | 106                          | 53  | 627               | 691  | 3294           | 2105 |
| Mn               | 145                        | 104 | 170       | 126 | 147          | 119  | 37                           | 18  | 167               | 168  | 1903           | 1515 |

<sup>1)</sup> number of samples

<sup>2)</sup> n for ash and organics/for minerals

<sup>3)</sup> g/kg

<sup>4)</sup> g/kg dry matter

<sup>5)</sup> g/kg dry matter

<sup>6)</sup> mg/kg dry matter

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## **Part D**

### **Role of nutrition on developmental diseases**



# Developmental diseases affecting growing horses

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## Abstract

There is a wide range of developmental diseases affecting growing horses. They have complex aetiopathogenesis, but are all primarily related to growth, nutrition and management. The list of conditions includes osteochondrosis, physitis, angular limb deformities, flexural deformities, tarsal bone collapse, wobblers disease and acquired vertebral deformities. Whilst there are many similarities between these conditions there are also important differences in the likely trigger factors, sites affected, onset and severity of clinical signs and outcome. Both the similarities and differences will be discussed in this paper. There has been considerable confusion over the terminology for this complex of conditions in young horses. It is suggested that a better term than Developmental Orthopaedic Disease (DOD) would be Developmental Skeletal Problems (DSP) as this does not imply the same aetiology or that the conditions principally involve bone. The incidence of these problems is high, probably between 10-50%, as indicated by different surveys. Many of these conditions can be treated by improved diet and efficient stud management. The challenges ahead are to improve early diagnosis, carry out more basic research to better understand pathogenesis and then to develop a better strategy for prevention.

*Keywords: developmental skeletal problems, growth, developmental orthopaedic disease, foals, young horses*

## Introduction

The problem of developmental diseases in young horses is huge and well beyond the scope of a single presentation. The aim of this paper is to set the scene for a series of more detailed presentations by giving a brief overview of the range of conditions involved. It is my intention to raise some issues that might later assist in providing a more evidence-based approach to developmental skeletal problems in foals that will ultimately result in a more effective scheme of prophylaxis and so reduce the overall incidence. These developmental diseases affect horses of all breeds and cost the horse industry worldwide many millions of euros each year.

## Defining the problem

Over the last 25 years much has been written about developmental conditions of foals, both in the scientific and lay literature. Despite all this a suitable definition for the overall problem has not been found. In the '60s and '70s each developmental condition was considered individually in its own right. With the increased interest in the conditions in the '80s, a meeting was convened by the American Quarter Horse Association (AQHA) in 1986. It was decided to collectively group all these conditions into a single category called "Developmental Orthopaedic Disease" (DOD) (McIlwraith, 1986). This was later challenged by Gabel (1988) who put forward an alternative term of "Metabolic Bone Disease" (MBD) because it implied a common pathogenesis. The reason behind this was because it was recognised that a foal could, on occasion, exhibit signs of all these developmental disorders

art the same time. I believe this supposition is incorrect and recent work shows how complex the pathogenesis is in some of the conditions (Jeffcott and Henson, 1998). If a foal has guttural pouch tympany and a dentigerous cyst at the same time it is not assumed that both conditions have the same pathogenesis. MBD also implies that only bone is involved which is certainly not the case with a number of the developmental conditions. Having said that it is logical to group these conditions as there are similarities between some of them and they all appear during the growth period. Perhaps a more appropriate title might be simply “Developmental Skeletal Problems” (DSP).

There has also been considerable confusion over the terminology of the specific DSP conditions as shown in Table 1.

*Table 1. List of developmental and skeletal conditions in foals and heir terminology.*

| Generally agreed term           | Synonyms                                                                                                                                    |
|---------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------|
| Osteochondrosis (OC)            | Osteochondritis dissecans (OCD)<br>Osteochondrosis dissecans<br>Dyschondroplasia (DCP)<br>Subchondral bone cyst (SBC)<br>Patellar dysplasia |
| Phyinitis                       | Epiphysitis<br>Physisosis<br>Physiolysis<br>Physeal dysplasia                                                                               |
| Angular limb deformities        | Bent legs                                                                                                                                   |
| Flexural deformities            | Contracted tendons<br>Ballerina syndrome                                                                                                    |
| Tarsal bone collapse            | Incomplete ossification of cuboidal bones<br>Cuboidal disease<br>Tarsal bone necrosis                                                       |
| Cervical vertebral malformation | Wobbler disease<br>Equine spinal ataxia                                                                                                     |
| Acquired vertebral deformities  | Kyphosis (Roach back)<br>Lordosis (Sway back)                                                                                               |

## General comments on developmental skeletal problems

The prevalence of these conditions in foals can be unacceptably high on some stud farms. Most breeds of horses are susceptible although ponies and feral horses seem to be much less commonly affected. The conditions all have complex aetiopathogenesis and these vary with the specific clinical entity. However, they are all associated with growth and are seen particularly in foals that are growing rapidly or not fed an adequate diet. The time of onset also varies (Table 2). The disease complex is believed to be associated with abnormalities of skeletal growth which begins with the conversion of cartilage to bone by the process of endochondral ossification.

*Table 2. Approximate time of onset of clinical signs and sites involved for Developmental Skeletal Problems.*

| Clinical condition       | Onset of signs | Sites involved                               |
|--------------------------|----------------|----------------------------------------------|
| Osteochondrosis          | 3-20 months    | Proximal and distal limbs and cervical spine |
| Phytitis                 | 24-8 months    | Distal limbs and stifles                     |
| Angular Limb Deformities | 1-6 months     | Distal fore and hind limbs                   |
| Flexural deformities     | 1-4 months     | Distal forelimb                              |
| Tarsal collapse          | 1-18 months    | Tarsal joints                                |
| Wobbler                  | 6-24 months    | Cervical spine                               |
| Vertebral abnormalities  | 6-9 months     | Thoracolumbar spine                          |

In addition to rapid growth and nutritional aspects other important features of pathogenesis include biomechanical stress on joint and growth cartilage from exercise, hormonal changes and a genetic predisposition. There are predilection sites for each of the conditions (Table 2) but in many instances multiple sites and multiple limbs can be involved (Figure 1).



*Figure 1. 7-month Thoroughbred foal that has had a rapid growth pattern and exhibits clinical signs of osteochondrosis of both hocks and stifles, phytitis of both carpi, flexural deformities of both forelimbs and acquired kyphosis of the thoracolumbar spine.*

### **What is the extent of the problem?**

The conditions in the DSP complex are essentially problems of management and diet, but they also appear to be relatively new diseases. For example, osteochondrosis (OC), which is the condition that has been written about most frequently, was first diagnosed in Sweden just after the 2<sup>nd</sup> World War (Nilsson 1947). It is not likely that clinicians would not have

recognised the fairly typical clinical signs of OC and pathologists would certainly not have missed the characteristic post mortem findings.

It is difficult to estimate the full extent of DSP in the horse population. There have been many incidence surveys based largely on clinical or radiological evidence. It is likely therefore that they are an underestimate of the real figure. The estimated prevalence of DOD has been reported as high as 50% (Knight *et al.*, 1985; O'Donoghue *et al.*, 1992) although this includes some conditions that are not necessarily considered to be developmental in origin. However, it is also clear that a balanced diet and affective stud management play a large part in reducing the incidence. Furthermore, many foals recover spontaneously and show no orthopaedic sequelae (Sandgren *et al.*, 1993; Aznam, 1994).

Another concern about DSP in the minds of many owners and breeders is to know whether the problem is progressively increasing over time. There are anecdotal reports on this, but no firm epidemiological data available at present. This raises the question of how important is the genetic involvement in pushing up the overall incidence. Apart from establishing that conditions like OC have some hereditary component there is no specific information. The paucity of accurate knowledge on genetic transmission is limiting the possibility of establishing a measuring programme of prophylaxis.

## **Diagnostic challenges**

Many of the DSPs are readily diagnosable by clinical observation and radiographic examination. However, early recognition is crucial as treatment and management of the problem are always much more effective in the early or mild stages of the conditions.

Careful clinical examination of foals and regular weighing to measure average daily gain (ADG) and monitor growth rate is particularly helpful. Many problems will be recognised in this way and by controlling growth rate and implementing proper mineral control can be overcome.

The other principal aid to diagnosis is radiographic examination which has been applied internationally to good effect (Falk-Rønne and Kristoffersen, 1980; Hoppe, 1984; Sandgren *et al.*, 1993; McIntosh and McIlwraith, 1993). However, in early cases the lesions may be confined to growth cartilage and no radiographic features are detectable. The use of ultrasound to estimate thickness of articular cartilage in the stifle joint in early cases of osteochondrosis may be valuable (Firth and Greydanus, 1987; Denoix, 1996; Predegast, 2001). The other technique that will become extremely useful when it is made widely available for horses will be magnetic resonance imaging (MRI) as both bone and soft tissue detail will be detectable.

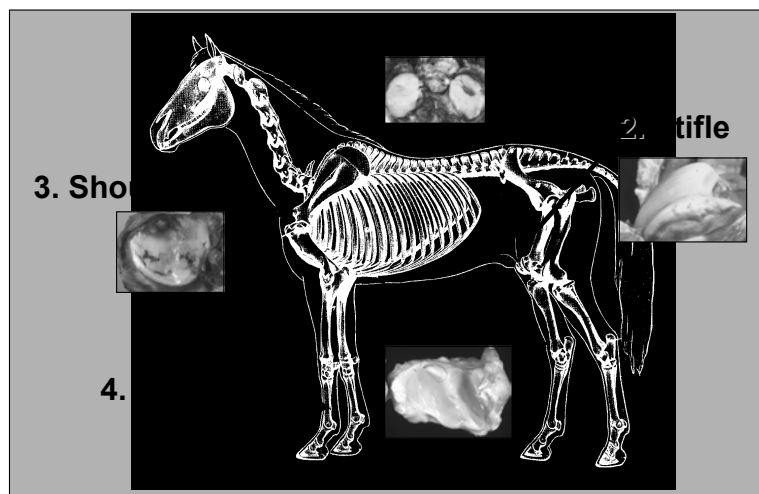
## **Developmental skeletal conditions**

### **Osteochondrosis**

Equine osteochondrosis is a developmental skeletal problem that costs the horse industry millions of euros each year (Jeffcott, 1991). The primary lesion in growth cartilage involves focal damage during the process of endochondral ossification. It occurs most frequently adjacent to the joint surface in the articular cartilage and results in the development of a



cartilage core that can then progress to subchondral bone damage, serious cartilage pathology and production of bony fragments (joint mice) (Figure 2).



*Figure 2. Predilection sites of osteochondrosis in horses.*

The clinical signs of equine osteochondrosis are difficult to characterise specifically because of the wide range of lesions and sites involved (Jeffcott, 1997). In severe cases, other signs of DSPs may also be present. The most common clinical sign of osteochondrosis is a non-painful distension of an affected joint (e.g. gonitis, bog spavin). Clinical signs may be divided broadly into two categories; those presented by foals usually under six months of age and those occurring in older animals. Often the first sign noted in foals is a tendency to spend more time lying down. This is accompanied frequently by joint swelling, stiffness and difficulty keeping up with other animals in the paddock. An accompanying sign may be the development of upright conformation of the limbs, presumably as a result of rapid growth. Fetlock osteochondrosis is particularly seen in younger animals (i.e. < 6 months of age) (Pagan and Jackson, 1996).

Marked lameness is not usually a feature of equine osteochondrosis, although it does occur with damage in some sites. For example, lesions in the shoulder frequently produce moderate to severe lameness, muscle atrophy and pain on joint flexion. In the stifle some cases of subchondral bone cysts in the medial femoral condyle present with lameness severe enough to suspect a fracture, but without a discernible site of pain or any joint swelling. The origin of pain in osteochondrosis presents a fascinating question. Horses often exhibit very marked pathological changes without showing much pain or distress. A very different situation from that observed in some other species and sites (e.g. canine elbow). The main signs in yearlings or older animals are stiffness of joints, flexion responses and varying degrees of lameness. These signs are usually associated with the onset of training and therefore suggest a biomechanical influence and an activation of sub-clinical or "silent" lesions.

Clinical diagnosis can often be made on the basis of signalment and signs. More definitive diagnosis requires the use of some specific clinical aids. Radiographic examination has been the traditional method of confirming diagnosis, but it should be remembered that early lesions involving cartilage without significant subchondral bone damage will not be visualised. In the distal limb oblique views may be helpful and in the hock as the commonest site of a lesion is

the distal intermediate ridge of the tibia the best view is a plantarolateral/dorsomedial oblique. Ultrasound examination of the swollen joints can also be helpful and is capable of showing up articular damage and joint mice. The most accurate way to confirm diagnosis is by arthroscopy (McIlwraith, 1990) and nowadays most of the predilection sites are accessible with the exception of the cervical articulations. Other aids include nuclear imaging (scintigraphy) which will usually have negative results unless there is active secondary bone damage. Magnetic resonance imaging (MRI) is a modality that would be ideal for diagnosis of both early and late lesions, but is currently unavailable to equine veterinary practice. Clinical pathology and the evaluation of synovial fluid may be helpful, but is used largely to eliminate inflammatory causes of swollen joints.

Although the initiating cause of osteochondrosis is unknown it is generally assumed to be multifactorial (Table 3). One of the most important keys to the whole problem is the ability to identify the first signs of a focal failure in the process of endochondral ossification. The primary lesion develops in growth cartilage and therefore a major area of investigation must involve the study of chondrocyte metabolism in growing foals (Figure 3). This will need to include molecular investigation of growth factor effects that are likely to have a direct effect on chondrocytes which may influence their metabolism. Insulin may have a role and the insulin-like growth factors and their binding proteins are profoundly affected by insulin states. The transforming growth factors (TGF) may well stimulate proliferation and the expression of the tissue inhibition of metalloproteinases thereby affecting the synthesis of extracellular matrix and collagen. The direct investigation of the chondrocytes themselves and the secretion of metalloproteinases and cysteine proteases is also important as they are capable of cleaving the components of extracellular matrix in preparation for angiogenesis and mineralization. Another important facet of the whole process is to understand more about the process of endochondral ossification. Work to identify certain markers (e.g. alkaline phosphatase, collagen type X secretion) has begun (Henson *et al.*, 1995; Henson *et al.*, 1996). From these studies it should be possible to go on and investigate the influence of other factors (e.g. copper, zinc, growth hormone, thyroid function) on growth cartilage metabolism. The future is hopeful and there is much to learn from the condition in other species, in particular poultry and pigs.

*Table 3. A list of some of the suspected aetiological factors in osteochondrosis in horses.*

- 
- Growth & body size
  - Nutrition
    - overfeeding
    - mineral imbalance
    - toxicity
  - Endocrinological involvement
  - Heredity / genetic predisposition
  - Biomechanics /exercise
- 

## **Phyistitis**

Phyistis is an abnormality of endochondral ossification in the metaphyseal growth plate where the structural integrity of the metaphyseal bone is weakened by the disturbance in maturation of the cartilage cells. This condition predisposes to microfracture and pain when the weight of the growing horse overloads the weight bearing capacity of the poorly structured metaphyseal bone. Microfracture cause formation of callus which is manifested clinically be characteristic

enlargement or flaring of the metaphysis of the affected long bones (White, 1980). Clinically, affected horses show metaphyseal flaring of the ends of the long bones particularly the distal ends of the tibia, radius, ulna and third metacarpal and metatarsal bones and proximal extremity of the front phalanx (Stashak, 1987). They may also show variable degrees of lameness, show an upright conformation at the fetlocks and are less active. Increased warmth and pain over the affected sites can be detected in severe and acute cases.

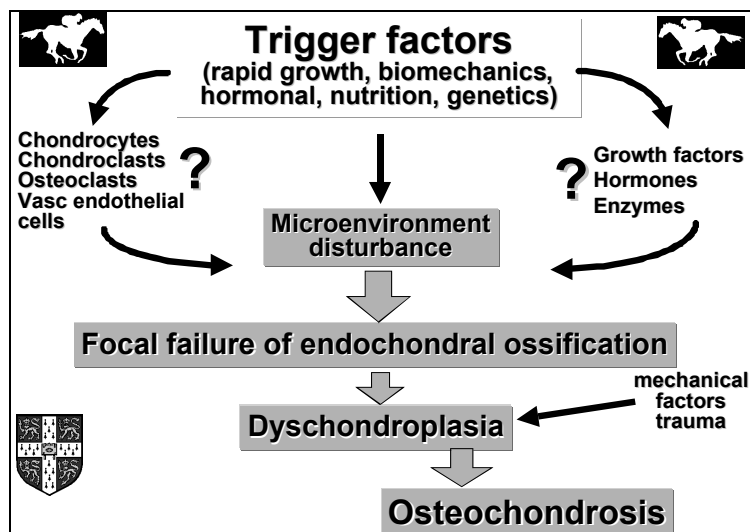


Figure 3. Simplified representation of a hypothesis for the pathogenesis of dyschondroplasia and osteochondrosis.

Phyinitis is probably the most frequently seen of the developmental skeletal problems in Thoroughbred foals. The condition is self-limiting and usually disappears as the growth plate closes (Turner, 1987). When the metaphyseal growth complex is involved, there is an interference of axial alignment of the limbs which affects the weight bearing ability of the limbs. Normal formation of the joint surfaces is disrupted when the epiphyseal growth complex is affected (Bramlage, 1987). Some studies (Arkin and Katz, 1956; Tschantz *et al.*, 1977) indicated that compression on the growth plate resulted in increased thickening of the physis due to retardation of provisional calcification and increased survival of chondrocytes.

Phyinitis may be concurrently associated with one of more types of developmental skeletal problems. A foal may at the same time show evidence of angular limb deformity (Turner, 1987), toe-in conformation (Rooney, 1963), flexural deformities (Moore and McIlwraith, 1977). Phyinitis and osteochondrosis were also reported in horses with wobbler syndrome (Mayhew *et al.*, 1978a).

The aetiology of phyinitis is not well understood. It is believed to be attributed to several interrelated factors, particularly high levels of nutrition that encourage rapid growth, trauma and the possible role of genetic predisposition although this last factor has not been proven. It appears that many aspects of phyinitis are possibly related to management because most affected horses are fast growers and are mostly fed high levels of nutrition, particularly high grain rations with an imbalance of mineral intake. Phyinitis may affect a few foals and if nutritional management has been the cause, there is a tendency for several other animals of

the same age group in the herd to be affected. The young, overweight and rapidly growing foals of less than 2 months of age are the most susceptible group. Peak incidence is normally reported after weaning when the foals are between 4-12 months old. The second phase when there is greater incidence usually occurred at the breaking season when the yearlings experience greater stress of being ridden and regularly exercised (Turner, 1987).

### **Angular limb deformities**

Angular limb deformities are attributed to one of the following defects: joint laxity, defective endochondral ossification of the bones in the carpus or tarsus, poor conformation, asynchronous longitudinal growth rate and traumatic luxation or fracture of the carpal bones (Leitch, 1979; Wagner and Watrous, 1991). These predisposing defects lead to uneven stress and growth at the metaphyseal growth plate which finally leads to the shifting of the normal limb axis.

Clinically, the condition is characterised by deviation of the limb from the sagittal plane in either a lateral or medial direction (Turner, 1987). If the portion of the limb distal to the affected area deviates away from the midline of the limb, it is referred to as medial or varus deformity. If it tilts toward the midline of the limb, the condition is called lateral or valgus deformity (Wyn-Jones, 1988). It is quite common to see foals born with some degree of deformity in one or more limbs, but it gradually disappears by about 2 weeks of age. Complete correction is possible if the deformities at the fetlock are surgically treated within 4 weeks and by 4 months if the carpus or tarsus is affected (Wagner and Watrous, 1991).

A variety of factors are suspected of being responsible for the development of angular limb deformities. These are the presence of immature bones in the carpus or tarsus, growth imbalance at the physis, laxity of the joint and trauma to the growth plate (Wagner and Watrous 1991). Imbalance growth of the growth plate of the ends of the long bones has been cited as one of the causes of limb deformities (Fretz, 1980). The growth imbalance can be traumatic in origin where the abnormal pressure causes one side of the growth plate to grow faster than the other which finally results in deviation of the normal limb axis. In fact, any factor causing continuous asymmetric pressure across the growth plate predisposes the occurrence of angular limb deformities. These factors include joint laxity, heavily muscled, vigorous and excessive activity, lameness or the opposite limb, growth imbalance at the epiphysis and defects in endochondral ossification of the cuboidal bones and small metacarpal bones. It is more common at the carpus than at the tarsus or distal end of metacarpus or metatarsus (Auer *et al.*, 1982).

### **Flexural deformities**

This is the condition affecting the limbs where the functional musculotendinous unit is relatively too short to maintain the normal limb alignment. However, the primary defect does not necessarily involve the tendon itself. The terms *flexural limb deformities* and *contracted tendons* have been used synonymously even though each conveys a different meaning. The affected foal shows fetlock flexion or hyperflexion.

Flexural limb deformities can be congenital or acquired and it is the latter form which can be regarded as a developmental skeletal problem. Deformities of the forelimb affecting the distal interphalangeal and metacarpophalangeal joints are the most common and cause contracture of the deep digital flexor and superficial digital flexor tendon respectively. Acquired flexural

deformities of the rear limbs affecting proximal interphalangeal and metatarsophalangeal joints are much less common (Wagner, 1990).

It is postulated that overfeeding and imbalance of nutrient intake during growth are responsible for the development of this deformity (Owen, 1975). It is further suggested by Owen (1975) that rapid growth with less exercise in foals causes a failure of tendons and ligaments to develop at the same rate as bone lengthening. However, the suggestion of discrepancy between bone and tendon growth has been largely unacceptable. Nevertheless, diet reduction and increased exercise have been commonly recommended with the clinical treatment (Von Matthiessen, 1993).

### **Tarsal bone collapse**

This condition is seen in young foals and is associated with collapse of the dorsal aspect of the central and third tarsal bones which causes a flexion deformity of the hock. Clinically there may be hock swelling, local pain and angulation (sickled conformation) of the hocks. There is not usually lameness at this stage, but stiffness of hindlimb gait with poor impulsion is common (i.e. bunny hopping). It is usually bilateral and can be confirmed by radiographic examination (Butler *et al.*, 2000). The tarsal bones appear smaller than normal and rounded due to incomplete ossification. There is irregular reduction in density and progressive collapse of the cranial aspect of the central and third tarsal bones with fragmentation.

The condition is often seen in neonatal foals but, may pass unrecognised until the horse starts work, when there may be an acute onset of hindlimb lameness or stiffness. At this stage secondary joint disease with collapse of joint spaces and periarticular new bone is present.

### **Wobbler disease**

Cervical vertebral malformation (CVM) of horses is characterised by stenosis of the cervical vertebral canal which leading to compression of the cervical spinal cord. In the young horse, dynamic stenosis (i.e. cervical instability) occurs from malformation of the dorsal facets of the cranial cervical vertebrae at the articulations of C<sub>2</sub>-C<sub>3</sub>, C<sub>3</sub>-C<sub>4</sub>, and C<sub>4</sub>-C<sub>5</sub>. Alternatively, there may be enlargement of the articular processes and/or soft tissues hypertrophy which causes static stenosis located caudally between C<sub>5</sub>-C<sub>6</sub> and C<sub>6</sub>-C<sub>7</sub> intervertebral spaces (Mayhew *et al.*, 1978a). This condition is common in 1-4 year old horses (Pool, 1993). The articular facets of the cervical vertebrae may have characteristic cartilage defects consistent with osteochondrosis (Wagner *et al.*, 1985; Reed *et al.*, 1987). The disease has been reported in light breeds of horses in North America and primarily occurs in 3-12 months old Thoroughbreds that are heavier than the average weight (Mayhew *et al.*, 1978a). The onset of CVM can be sudden or slowly progressive (Wagner, 1982). Affected horses develop incoordination of the hind or all limbs and shows a typical wobbling gait (Mayhew *et al.*, 1978a).

There has been a strong suspicion over the years that CVM is an inheritable condition (Prickett, 1968; Falco *et al.*, 1976), but evidence from epidemiological studies do not support this view. However, Cain (1993) suggested that wobbler horses are born with relatively narrow spinal canals and combined with a rapid growth rate cause malalignment of the canal. With lesser space, the spinal cord could easily be compressed. Studies by Donawick *et al.* (1989) showed an important relationship between over nutrition and incidence of CVM in the young and fast growing Thoroughbred horses. Combining overfeeding and excessive exercise

in young horses may induce necrosis at the bone/cartilage junction of the articular processes of the vertebrae. The continuous growth of vertebral cartilage without ossification leads to stenosis of the vertebral canal and compression of the spinal cord (Donawick *et al.*, 1989). This relationship has been previously suggested in horses (Hintz and Schryver, 1976) and Great Dane dogs (Wright *et al.*, 1973; Hedhammar *et al.*, 1974). It has also been demonstrated that dietary restriction and resting young horses with early signs of CVM prevent cervical vertebral abnormalities (Mayhew *et al.*, 1978b). This measure allows time for the vertebral canal diameter to enlarge and reduce the compression on the spinal cord. However, if left untreated and continued to be overfed and vigorously exercised, CVM progresses rapidly and the prognosis is poor.

### **Acquired vertebral abnormalities**

These conditions are much less frequently seen than the previous categories, but are also associated with rapid growth. The most common is kyphosis (roach back) that occurs post-weaning usually from 6 to 9 months of age. The upward curvature of the caudal thoracic and lumbar spine occurs quite rapidly. If detected soon enough and the growth rate is cut back then there may be improvement. In most cases the degree of kyphosis remains constant and the animal will be left with a rigid spine and susceptibility to back problems.

The other developmental vertebral abnormality is lordosis (sway back) which is often a congenital problem that can be exacerbated as the foal grows. The problem is more common in Saddlebred horses than other horses.

### **What are the challenges ahead?**

I see the principal challenge for the future of DSPs is to reduce their incidence and limit the economic “wastage” to the horse industry worldwide. Despite the considerable knowledge achieved in recent years there are still many gaps and unanswered questions. The problem needs to be studied *in toto* and with a multidisciplinary approach. For example:

- There have been no prospective epidemiological studies to assess the prevalence of the overall problem and to objectively understand the important influences of nutrition and stud management.
- Evidence based field studies providing significant differences between the outcomes from study and control groups to evaluate the effects of introducing improved feeding and nutritional regimes on studs.
- The underlying pathogenesis of all DSPs is still crucial to the future of the horse breeding industry. This will inevitably require further research into endochondral ossification. It is undoubtedly due to faults in the complex process during early development that many of the DSPs arise. As far as OC is concerned there are a number of research groups around the world making valuable contributions (Jeffcott and Davies, 2000). The Utrecht group led by van Weeren and Barneveld (199) carried out a very important multidisciplinary study that has greatly increased knowledge on the development of OC.
- The role of copper and other trace minerals and their influence on the prevalence of DSPs is crucial as there is so much that we do not understand. The excellent work carried out in New Zealand (Pearce *et al.*, 1998) demonstrates that much more needs to be done to properly understand the role of copper in endochondral ossification and the induction of DSPs (Jeffcott and Davies 1998).
- Another exciting area for investigation is to better understand the role of high energy feeding of young foals and the resultant high incidence of OC (Savage *et al.*, 1993),

hyperglycaemia and hyperinsulinaemia (Ralston, 1996). We also know that insulin has an effect on maturing chondrocytes in the growth plate (Henson *et al.*, 1997).

- The role of biomechanics/exercise needs further indepth research. Field studies showed apparent beneficial effects of exercise in reducing the clinical signs of OC (Bruin and Creemers, 1994) although this was not really confirmed by the Utrecht study (Barneveld and van Weeren, 1999). Recent work in Cambridge using an *in vitro* model (Bowe, 2003) is indicating that impact loading affects chondrocyte morphology and initiates an alteration in the expulsion of metalloproteinases (MMPS) within chondrocytes. This could have important implications in endochondral ossification.
- The other area that very little work has been done so far is to better understand the genetic background of DSPs. Apart from some work in Scandinavia on Standardbreds and Warmbloods that is very little published in this important area.

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# The potential impact of nutrition on bone growth in horses

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## Introduction

Bone formation is an extremely complex physiological process taking place at three centres of 'endochondral ossification' - the epiphysis, the metaphyseal growth plate and at ossification centres inside non-longitudinal bones. The process as mentioned before is initiated by chondrocytes from cartilaginous growth plates within these areas (Anderson, 1990). Chondrocytes can be divided into two types - those which are destined to remain at the articular joint surfaces, protecting the bone and allowing for movement, and those which will be completely calcified (= ossification) to form new bone (e.g. longitudinal bone growth) (Hurtig and Pool, 1996). Within the articular-epiphyseal complex both mechanisms take place to create the enlarged rounded 'head' of the bone, which supports the articular cartilage of the joint. Figure 2.1 below shows the location of the process and gives an example of a lesion (Van Weeren and Barneveld, 1999b). The process can be described sequentially along the merging zones and has been discussed in detail by a previous paper within this series. Figure 1 below summarises the process once more.

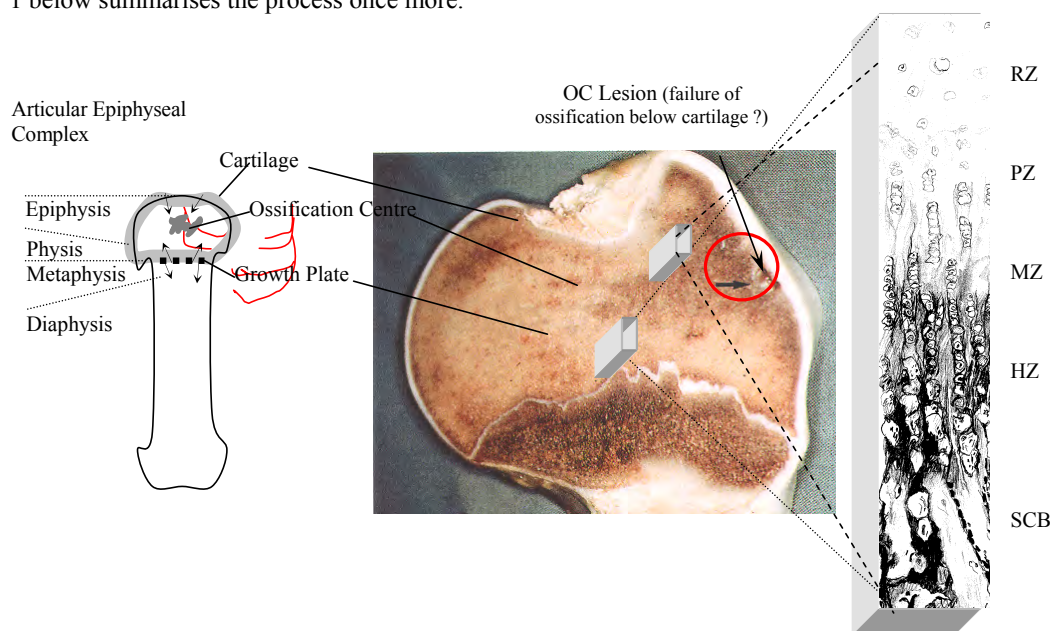


Figure 1. Chondrocytes 'develop' along zones, starting at the chondrocyte resting zone (RZ), where the growth plate begins, followed by the chondrocyte proliferation zone (PZ), leading into the maturation zone (MZ) - also called upper hypertrophic zone, moving on into the hypertrophic zone (HZ) which converts into subchondral bone (SCB) (\*Photo: Van Weeren and Barneveld, 1999b).

Previous nutritional research has concentrated on isolating nutrients, excess or deficiency of which may lead to increased probability of developing the condition. These hypotheses were then tested *in vivo*, and if proved correct led to an induction/increase of lesions. Some excellent reviews concentrating on the link of osteochondrosis and nutrition in other animals have been published. Both in pigs and dogs this condition has been researched extensively in 'in vivo' experiments (Wolter, 1996; Richardson and Zentek, 1998; Schoenmakers *et al.*, 2000).

The interrelationship between varying factors which have a negative impact on healthy bone development is not yet explained and many questions must still be answered:

- What nutritional elements are involved in the bone formation process?
- How does nutrition affect other contributory factors, such as endocrine changes?

This paper is taken from an extensive report (Ellis, 2001), where a detailed bibliography is given, and practical implications as well as further research are further discussed.

In order to understand the potential influence of nutrition on bone growth in horses, this paper gives a more detailed biochemical overview of the process and related nutritional substances as it is understood at present. Detailed identification of nutritional factors, which are implicated in the various metabolic phases of bone formation and mal-formation, will be highlighted. Appendix 1 gives a list of abbreviations and a glossary of some terms used.

It must be pointed out that much of the current knowledge of the growth process at metabolic level has been gained from other mammal species, while limited research in horses exists thus far.

## **The process of bone formation from a nutritionist's point of view**

### **The synovial joint**

The articular surface of the newborn foal is already lined with a soft protective layer of cartilage. Unlike some mammals, cells in the equine articular cartilage are not yet organised into stacks, but randomly floating in the matrix (Jeffcott and Henson, 1998). When looking at the growing process at the articular-epiphyseal plate, it is unclear at which point chondrocytes differentiate into those which maintain the elastic, protective joint cartilage at the upper end of the bone, and which ones become hypertrophic, die and form part of the subchondral bone layer. Influences of the synovial fluid on the process have only recently been investigated in horses.

Interleukin -1 produced by synovial cells stimulates the production of collagenase and caseinase, capable of destroying cartilage tissue (Spiers *et al.*, 1994). Spiers *et al.* (1994) also found that equine neutrophils (essentially white blood cells, travelling to sites of inflammation) produce destructive proteinases, possibly contributing to the pathology of joint disorders.

### **The resting zone**

In the resting zone, uniform cells lay 'dormant'. This zone is also called the germinal or reserve stem cell layer zone and cells have a phenotypical appearance of fibroblasts (Isaksson *et al.*, 1987). Some of these cells are 'dormant chondrocytes' or pre-chondrocytes waiting to be activated. The enzyme Cathepsin B plays a role in intracellular digestion of the

chondrocytes remaining at the articular surface and it is believed that under pathological conditions, which may or may not have been initiated from below (- the hypertrophic zone), the release of this enzyme (making it extracellular) can lead to degradation of prostaglandins, aggrecans, link proteins and also collagen types I (bone collagen), II, IX and XI (Mort *et al.*, 1984; Maciewicz *et al.*, 1991; Sires *et al.*, 1995). Hernandez-Vidal *et al.* (1998) found an abundance of the enzyme in chondrocyte clonal clusters adjacent to equine OC-lesions. Although it is generally assumed that lesions are a symptom of subchondral bone failure, which may well lead to the release of Cathepsin B, the reverse may be the case. A possible influence of nutrition (possibly proteins), related growth hormones, and minerals or a genetic mal-function have not yet been investigated. Davies *et al.* (1996) demonstrated that Copper can affect the levels of cathepsin B in cultured cartilage.

Although the exact trigger of differentiation has not been fully defined, it is known that growth factors and hormones stimulate chondrocyte proliferation (Hochberg *et al.*, 1989; Hoskins and Asling, 1977; Isaksson *et al.*, 1987). Apart from the genetic pre-disposition towards growth hormone secretory capacity and insulin like growth factor levels, which has been fully established in dogs (Eigenmann *et al.*, 1984; Wolter, 1996), nutrition has also been directly linked to production levels of these substances both in humans, horses, dogs, pigs and other species (Richardson and Zentek, 1998; Danforth and Burger, 1979; Daughaday *et al.*, 1975; Froesch *et al.*, 1985; Hedhammer *et al.*, 1974; Scott *et al.*, 1996).

### **The proliferative zone**

As stem cells differentiate they enter the proliferative zone, where they become enlarged and multiply at a limited rate. Here they start organising themselves into a columnar arrangement towards the hypertrophic zone. Vitamin-A has been identified as stimulating chondrocyte differentiation at this stage (Wu *et al.*, 1996; Orth, 1999). Deficiency of this substance will lead to a poorly differentiated growth plate and toxicity will lead to a prematurely closed growth plate, as the Vitamin can 'over-stimulate' matrix calcification at a later stage (Orth, 1999, Soeta *et al.*, 1999). Again hormones and growth factors have been isolated as regulatory to this process, such as thyroxine, transforming growth factor- $\beta$  (TGF- $\beta$ ), basic fibroblast growth factor and insulin growth factor - 1 (IGF - 1) (Orth, 1999).

Overnutrition can induce increased hormone production, which will lead to excess proliferation of chondrocytes at this stage, determining initial fast growth. Increased growth rates have been directly implicated with osteochondrotic lesions in other species and some links have been made in horses (Hochberg *et al.*, 1989; Glade *et al.*, 1983; Glade and Belling, 1984). Henson *et al.* (1997a) have recently investigated IGF-1 and 2 in equine foetal chondrocytes as well as the effect of insulin on these. Increased production of insulin can follow hyperglycaemia after excess grain ingestion in horses. Ralston (1996) found that horses with OC had higher postprandial glucose and insulin responses to feeding than normal horses, thus being metabolically (genetically or through nutrition?) predisposed to excess insulin production. However, this was an early limited study which needs to be repeated with a more homogenous group of horses and exact hormonal mechanisms must be determined.

Throughout the zones cells are suspended in extra cellular matrix (ECM), partially produced by the chondrocytes themselves, containing water, hyaline, proteoglycans (PG's), keratin sulphate, enzymes and minerals. These are in the form of more complex molecules, such as various collagens, aggrecans and fibronectins. Epiphyseal arteries provide proliferating

chondrocytes with necessary nutrients such as oxygen and growth factors (Henson *et al.*, 1996).

The supply of minerals and vitamins acting as co-factors for enzymes is partially dependent on correct provision of nutrients for absorption into the blood in the digestive tract. Chondrocytes release enzymes such as the Metalloproteinases (MMP's). These break down collagen and gelatin during later stages (to be replaced by mineralised tissue). MMP's are key-regulators of the mineralisation process and are known to be zinc-dependent (Coughlan *et al.*, 1998). When released by the chondrocytes MMP's are inactive and need to be activated or in turn inhibited by localised (tissue inhibitor) enzymes (TIMP's). Ward *et al.* (1991) have isolated one MMP (pro-MMP2 = a TIMP) in humans and also found this to be calcium dependent. More recently, Henson *et al.* (1995, 1996), Clegg *et al.* (1998) and Brama *et al.* (1998, 2000) have confirmed the presence and actions of many of the above components in equine growth cartilage. Brama *et al.* (1998) found no change in MMP activity between normal and osteochondrotic foals at 5 and 11 months but confirmed that foal MMP action is almost double that of adult horses.

### Upper hypertrophic zone

In order to provide the necessary building blocks for mineralisation chondrocytes have accumulated inorganic  $\text{Ca}^{2+}$  (Wuthier, 1977 and 1982). This is linked to inorganic phosphate (Pi) and phosphatylserine (Ca-PS-Pi) (Whuthier, 1992). The minerals found in the mitochondria are not yet crystalline but are in an amorphous calcium phosphate form which is stabilised by the presence of ATP (adeno-tri-phosphates) and  $\text{Mg}^{2+}$  which prevents the formation of apatite (the crystallised form of calcium phosphate). At this stage the ECM must be kept pliable and soft and proteoglycans are believed to inhibit too early mineralisation in the ECM (Buckwalter *et al.*, 1987; Dziwiatkowski and Majznerski 1985, Blumenthal *et al.* 1979; Hunter, 1996). A reduced proteoglycan metabolism in osteochondrotic articular cartilage of foals has recently been reported, making such chondrocytes less 'vital' (Van den Hoogen *et al.*, 1999).

As chondrocytes mature they form into orderly columns - longitudinal septae, which provide a scaffold for the increasing extracellular matrix, becoming denser in structural cartilaginous compounds. Density is increased by the release of matrix vesicles (MV's), and the predominant type II collagen. Matrix vesicles originate from hypertrophic chondrocytes, by being released from their plasma membrane containing the accumulated calcium phosphate from the mitochondria (Bonucci, 1970; Kirsch *et al.*, 1997; McLean *et al.*, 1987; Wuthier, 1982; Henson *et al.*, 1995). MV's are rich in lipids, containing acidic phospholipids (phosphatidylserine, phosphatidylcholine) with an alkaline phosphatase (a zinc-dependent MMP) acting upon those to produce inorganic phosphate for the mineralisation process (De Bernard *et al.*, 1977).

Phospholipids in chondrocytes, matrix vesicles and the ECM are vital to create the correct environment for enzymes and cell characteristics (fluidity, permeability - transport). These are regulated by a Vitamin D derivative hormone (calcitriol) both at the endochondral growth plate as well as in the digestive system (Boyan *et al.*, 1992). Two Vitamin D metabolites are locally produced by chondrocytes under hormone and GF regulation (Boyan *et al.*, 1992). Results of the recent osteochondrosis study in foals at Lelystad, showed that phospholipid content was significantly increased in osteochondrotic subchondral bone (Van de Lest *et al.*, 1999b). The ratio between the different lipids had also changed. In rats and humans essential

fatty acid deficiency has had a direct impact on dental and bone development (Gilder and Boskey, 1990). Kirsch *et al.* (1997) report on the importance of the lipid composition for the accumulation of  $\text{Ca}^{2+}$  into matrix vesicles mediated by Annexin V formed from phosphatylserine (Rojas *et al.*, 1992). Annexins have a  $\text{Zn}^{2+}$  binding site, and Sauer *et al.* (1989) report of an inhibition of the Annexin  $\text{Ca}^{2+}$  uptake through excess zinc.

MV's start forming intraluminal crystals of hydroxyapatite (HAP) and release collagen type II, VI and X molecules (McLean *et al.*, 1987; Wu *et al.*, 1996; Kirsch *et al.*, 1997; Jeffcott and Henson, 1998). High levels of  $\text{Mg}^{2+}$  within the soluble fraction of MV's effectively prevent conversion of  $\text{Ca}^{2+}\text{Pi}$  to crystalline mineral and a chemical equilibrium between various ions, such as  $\text{CO}_3^{2-}$ ,  $\text{Mg}^{2+}$ ,  $\text{Na}^+$ ,  $\text{F}^-$  and pyrophosphate influence the final properties of the crystal formed (Wuthier, 1982). Wuthier (1982) concludes that part of the crystallisation process is the enzymatic degradation of the MV's membrane to allow for leaking out of the electrolyte ions like  $\text{Mg}^{2+}$ . Wu *et al.* (1996) have recreated the nucleationally active core of matrix vesicles from in vivo extraction in order to analyse the rate of calcium uptake and the effect of other elements on the process. Inclusion of Annexin V led to increased activity, and when adding low levels of zinc and magnesium a more rapid  $\text{Ca}^{2+}$  uptake was exhibited. At higher levels, however,  $\text{Ca}^{2+}$  uptake was significantly delayed (Wu *et al.*, 1996).

Cadmium has also been identified as an Annexin inhibitor (Chvapil and Misiorowski, 1980; Kirsch *et al.*, 1997). This emphasises that despite nutritional research concentrating on deficiencies or excess of certain individual elements, correct mineral balance and ratios are most likely of utmost importance.

As MV's become saturated with HAP crystals they break up releasing their contents into the extra cellular matrix (Buckwalther *et al.*, 1987; Scherft and Moskalewski, 1984). The calcification of the matrix begins and proteoglycans are believed to play a vital role at this stage. Together with the MMP's who are no longer inhibited by TIMP's, they break up the more elastic collagens (II, VI) to allow for the deposition of HAP crystals and the formation of type I Collagen.

Hypotrophic chondrocytes undergo apoptosis (programmed cell death), leaving behind a columnar arrangement of debris and attached particles (collagens, ECM -HAP's). Insulin, in vitro, acts as a 'survival factor' preventing chondrocyte apoptosis (Henson *et al.*, 1997a). The ECM becomes highly concentrated with aggregating cartilage proteoglycans, type II collagens, newly synthesised type X collagen and alkaline phosphatase. In avian dyschondroplasia a Vitamin D<sub>3</sub> deficiency has been implicated with reduced type X Collagen production by hypertrophic chondrocytes (Xu *et al.*, 1998). However, Henson *et al.* (1996) concluded that unlike bovine, ovine or canine type X collagen, equine X collagen is not disulphide bonded, illustrating that not all mechanisms discovered in one species can be assumed in horses.

PG's, which earlier inhibited mineralisation, have now changed shape (Scherft and Moskalewski, 1984; Alini *et al.*, 1992). They are reduced in size and are no longer attached to hyaluronic acid strands, which have become free in the ECM (Buckwalther *et al.*, 1987). Initially a deposition of hydroxyapatite crystals on type II collagen takes place. This collagen is degraded into crystallised hydroxyproline chains which integrate with the type X collagen and proteoglycans to form part of the newly calcified longitudinal septae (Alini *et al.*, 1992; Wuthier, 1982). In other species the removal of the thyroid gland has led to a failure in initiation of this last stage (Glade and Belling, 1984 and 1986; Jeffcott and Henson, 1998).

Therefore, the link to thyroxine and its effects on sub-chondral bone formation is confirmed. The thyroid hormones ( $T_3$  and  $T_4$ ) are also involved in metaphyseal invasion of growth cartilage by blood vessels, and these in turn are influenced by insulin.

### Subchondral bone

New capillary sprouts/buds invade the subchondral area from below supplying further necessary nutrients to complete formation of sub-chondral bone. In horses, type VI collagen has an interesting role, being concentrated along hypertrophic chondrocytes, where capillary buds are invading, possibly as a strengthening matrix (Henson *et al.*, 1996). These cartilage canal vessels have been shown to disappear both in horses and other mammals after a certain age, partially explaining the 'window of opportunity' to harm or improve correct nutrient supply in the young foal. In pigs a defect in cartilage canal blood supply has been repeatedly reported as a very possible cause of OC lesions (Kincaid *et al.*, 1985; Carlson *et al.*, 1989).

Equine cartilage canal vessels become necrotic from the age of 4.5 months, depending on site, and disappear from all sites by the age of 7 months (Carlson *et al.*, 1995). Carlson *et al.* (1995) also found that cartilage canal vessels were necrotic or missing in areas of OC lesions of 5 months old foals ( $n=5$ ). Such a vascular defect could be a secondary symptom of trauma or caused by a nutritional deficiency (Shingleton *et al.*, 1997). Glade and Belling (1986) link a lack of capillary penetration to possible inhibitors, such as glucocorticoids and a phosphate-Vitamin D deficiency (see 3.4.1). When glucocorticoids were administered via dexamethasone to mares and foals intra-venously, a failure of energy provision for the synthetic bone formation pathways due to a lack of blood supply was observed (Glade *et al.*, 1981). This may have been due to a failure in presenting a 'recognition signal' to the capillary buds by the hypertrophic chondrocytes or a possible inhibitor.

Along with the blood supply osteoblasts and osteoclasts are deposited along the calcified septae. The osteoclasts secrete cathepsin B and L which resorb bone and soft cartilage, completing the type II and VI degradation, while osteoblasts support the forming of type I collagen ossified bone (Lee *et al.*, 1995). Lysyl oxidase, a pituitary hormone-dependent enzyme plays a major role in cross-linking the lysyl residues of collagen and elastin into covalent cross-links (Seigel *et al.*, 1970; Shoshan and Finkelstein, 1976). This enzyme is known to be copper dependent and zinc can attach to the Cu-binding site and inhibit its function (Chvapil and Misiorowski, 1980). Van de Lest *et al.*, (1999b), found an increased lysyl oxidase activity at sites of equine osteochondrotic lesions.

Phospholipids still play an important role in the continuing mineralisation process and excess amounts of phospholipids at this stage (as found in equine OC lesions, Van de Lest *et al.*, 1999b) may lead to a disturbance of the  $Ca^{2+}$ -phospholipid-phosphate equilibrium, preventing proper mineralisation (Van de Lest *et al.*, 1999b). Lillich *et al.* (1997) also found that there was a decreased level of uronic acid, glycosaminoglycan and chondroitin sulphate in cartilage of OC positive joints compared to normal cartilage from the same area.

At this point the subchondral bone composition is around 65% hydroxyapatite crystals, 35-40% water and 10% collagen type I (Brama, 1999). Osteoblasts differentiate to release the Vitamin K dependent protein osteocalcin under Vitamin D regulation, continuing and finalising the ECM mineralisation process (Stein *et al.*, 1992).



## Hormonal regulation of cartilage growth and metabolism

The process of growth takes place under endocrine control, as may have become clear in paragraphs 2.1-2.5 (Lebowitz and Eisenbarth, 1975). Key hormones involved and influenced by nutrition are the insulin like growth factors (IGF's), also called somatomedins, and the thyroid hormones – thyroxine ( $T_3$ ) and tri-iodothyronine ( $T_4$ ) (Phillips *et al.*, 1974). The parathyroid (PTH) hormone also plays an important role during the calcification process together with the Vit-D hormone Calcitriol.

Growth hormone (GH) regulation is mediated by insulin-like growth factors (Nap, 1993). Receptors of IGFs are present in most cells. They are divided into IGF type I and IGF type II. Type I receptors are mainly present in fibroblasts, chondrocytes and osteoblasts (Thissen *et al.*, 1994). IGF's stimulate glucose transport as well as glucose protein and lipid synthesis within target cells. They are potent growth promoters for many different cultured cells, by stimulating DNA synthesis and cell replication in vitro (Froesch *et al.*, 1985). IGF's are regulated by the hypothalamus through growth hormones and become responsive to changes in nutrition after birth. Overstimulation of this organ through excess energy intake has shown a marked increase in circulating IGF's following excess glucose absorbed into the blood – leading to increased growth rates in pigs, dogs, horses and rats (Ashton and Francis, 1978; Phillips *et al.*, 1979; Isaksson *et al.*, 1984; Froesch *et al.*, 1985; Henson *et al.*, 1997a). Insulin factors are important for chondrocyte survival, by suppressing differentiation and later apoptosis during in vitro experiments with IGF-I and II on neonatal chondrocytes (Henson *et al.* 1997a). Otte *et al.* (1996) cloned and sequenced equine IGF-I cDNA, finding its nucleotide homology close to the human equivalent (96.3%). There was no difference between foetal and adult tissue. That growth hormones have a direct influence on zinc, copper, manganese and selenium metabolism has been established in human research (Aihara *et al.*, 1985).

Recently the genetic regulation of the IGF system has been investigated by Rhoads *et al.* (2000), who showed that an 'acid labile sub-unit (ALS) gene' expressed in the liver produces an ALS, which together with GH determines the maturation of the IGF system. Rhoads *et al.* (2000) found that in sheep ALS production and final maturation of the IGF system was delayed following undernutrition during late foetal and early post-natal life, with the opposite effect for ad libitum fed animals. Maternal over-nutrition may lead to a faster maturation of the GH-IGF axis (Boisclair, 2000).

Thyroid hormones act in conjunction with the growth hormones. Daughaday *et al.* (1975) reviewed an experiment in rats, in which thyroxine alone stimulated some bone growth, but a significant increase in epiphyseal cartilage growth was recorded when thyroxine acted in conjunction with growth hormones. Thyroxine is converted to tri-iodothyronine by the selenoenzyme iodine thyronine-5'. McLaughlin and Doige (1982) report that neonatal foals have higher thyroid hormone values than any other species. These authors investigated the ossification stages of pre-mature, neo-natal and thyroidectomised foals. Results showed that equine carpal and tarsal bones are particularly susceptible to hormonal manipulation during the last months of gestation. Thyroidectomised foals had a highly decreased rate of ossification.

Growth hormones are also influenced by many other factors, and interestingly oestrogens, have repeatedly been mentioned in the literature. Oestrogen plays a role in adult bone homeostasis by inhibiting chondrogenesis and the growth of bone tissue (Goedegebuure and Hazewinkel, 1981; Jansson *et al.*, 1983). Schwartz *et al.* (1969) report that oestrogen given to

young rats and chickens inhibited growth of bones and depressed spontaneous epiphyseal cartilage growth in rabbits. In sheep, ingestion of estrogenic compounds through red clover pastures and silages have been reported to have similar effects (Kallella, 1973; Nwanna *et al.*, 1995).

Changes in hormone production and metabolism may be due to nutritional, genetic or other factors. Henson *et al.* (1997b) found that transforming growth factor- $\beta$  (TGF- $\beta$ ) expression and localisation was altered in dyschondroplastic equine growth cartilage, pointing towards a genetic pre-disposition. This may be breed linked. In dogs, an apparent correlation between body size and IGF-I levels has been established in vivo (Eigenmann *et al.*, 1984). Lower IGF-I levels in plasma could also be observed in osteochondrosis positive foals (Sloet Van Oldruitenborgh-Oosterbaan *et al.*, 1999). Whether these changes are possibly primary causes of, or a response to localised lesions is still unclear. If proven as causative, the origin of these changes could lie in genetic programming, nutritional imbalance or other factors. Sloet Van Oldruitenborgh-Oosterbaan *et al.* (1999) did not find any correlation between different exercise levels with these results. However, little is known about the connection between plasma IGF levels at the subchondral bone. The relationship of plasma IGF levels and their production at growth plate cartilage is unclear.

## Nutritional elements and bone formation

The previous chapter showed how the development and growth of bone in the foal involves many metabolic substances (e.g. enzymes, hormones), which are dependent on 'building blocks' (carbohydrates, minerals, amino acids, vitamins) provided directly via nutrition or influenced by nutritional supply. These can be crudely divided into carbohydrates, proteins, lipids and minerals.

The summary of the main regulators of bone-resorption and bone remodelling in the adult animal in Table 1 gives some indication as to the regulation of bone growth in the foal and HOW most of these substances have or may have a direct or indirect link to nutritional supply:

## Conclusion

From this short overview, it can be concluded that nutrient composition of feed has an important influence on the bone development of the foal. Even feeding level and composition of feed of the mare may influence genetic programming of foetal development. On the other hand, the **relative** importance of individual nutritional influences is still not fully understood:

- Many links are circumstantial or are based on knowledge from other animals.
- The supply of minerals and vitamins acting as co-factors for enzymes is partially dependent on correct provision of nutrients for absorption into the blood in the digestive tract.
- Much of the basic knowledge in mineral metabolism and on hormonal regulation in the mare and foal has yet to be researched.
- The link between absorption of feed substrates and hormonal regulation of bone formation and growth needs to be explored further.

At the current level of knowledge pure experiments concentrating on developmental diseases in relation to one individual nutrient would be necessary, but such research would need to be extremely extensive in order to compensate for the many variables (i.e. – unknown actions and interactions of other nutrients).

Table 1. Main substances involved in the bone formation process and possible nutritional links.

| Substance             | Function                                                                                                                                                                | Nutritional Link                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         |
|-----------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Annexin V             | regulator                                                                                                                                                               | Magnesium has been identified as an activator of many enzymes. In bone formation it plays a role in the Annexin V regulation of $\text{Ca}^{2+}$ uptake into Matrix Vesicles and like zinc, Mg plays a vital role at low levels, which can become detrimental at higher levels, by binding to Cu-attachment sites (Chvapil and Misiorowski, 1980; Kirsch <i>et al.</i> , 1997). In the hypertrophic chondrocyte and later the MV's magnesium prevents premature crystallisation of $\text{Ca}^{2+}\text{Pi}$ . Magnesium is therefore closely related to the Ca and P metabolism and is also resorbed from the bone during a deficiency. |
| Calcitonin            | reduces osteoclast activity, increases osteoblasts                                                                                                                      | An excess of Ca stimulates secretion of another hormone, Calcitonin, which actually decreases osteoclastic bone-resorbtion, it also increases osteoblast (mineral-depositing) activity (McGilvery and Goldstein, 1983) and it reduces the loss of Ca and phosphate ions in the urine. Furthermore, phytate absorption is influenced negatively by excess dietary Ca with disproportionate low levels of P (Schoenmakers <i>et al.</i> , 2000).                                                                                                                                                                                           |
| PTH                   | inhibits chondrogenesis, bonegrowth                                                                                                                                     | Excess phosphorus intake will stimulate PTH to reduce P absorption, which in turn will increase Ca absorption and together with calcitriol will accelerate bone absorption.                                                                                                                                                                                                                                                                                                                                                                                                                                                              |
| Vitamin A             | cartilage maturation, bone resorbtion, collagen synthesis                                                                                                               | b-caroteen - obtained from fresh forages and converted to Vit A                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          |
| Vitamin C             | proliferation and maturation of chondrocytes                                                                                                                            | Synthesized, Deficiency unlikely, slight indication that extreme excess may be harmful                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   |
| Vitamin D metabolites | 1,25-(OH) <sub>2</sub> Cholecalciferol:                                                                                                                                 | stimulate Ca and P absorption<br>Lack of Vitamin D or Calcium reduces renal absorption of P and can cause a delay in growth and closure of epiphyseal plates.                                                                                                                                                                                                                                                                                                                                                                                                                                                                            |
| Growth hormones       | regulation of growth rates of bone chondrogenesis, osteogenesis                                                                                                         | Soluble carbohydrates absorbed from diet – affect Insuline production rate/ balance<br>Iodine is converted to iodide in the gut and absorbed into the blood stream, from which it is actively transported to the thyroid gland, to form an essential component of the thyroid hormones T <sub>4</sub> and T <sub>3</sub> . Iodine deficiency leads to stunted growth, through a compensatory hyperthyroid reaction while iodine excess can lead to reduction in thyroid activity.                                                                                                                                                        |
| Thyroxin              |                                                                                                                                                                         |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          |
| Glucosamine           | mucopolysaccharides, which form the major components of cartilage (chitins, chondroitins) and the amino-monosaccharide glucosamine (GAG's, hyaluronan, keratan sulfate) | In humans a strong role for these agents in symptom management for osteoarthritis has emerged but no curative effect can be proven (Deal and Moskowitz, 1999). It needs to be assessed whether these substances, if taken orally, are absorbed and whether they reach the areas they purport to 'help' in horses. Whether these supplements can lead to increased repair or prevention of, osteochondroitic lesions, certainly may warrant further research, following the current understanding of their bio-availability and actions.                                                                                                  |
| Oestrogen             | increases osteoclast bone resorbtion                                                                                                                                    | Can be produced by moulds on badly stored forages; found in some moulds in clover fields;                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                |

The nature of this paper was extremely theoretical but for the practising horse breeder it is of utmost importance to gain clear knowledge on how to create the best possible conditions for prevention or resolution of developmental diseases in foals. Nutrition is one important factor, which can actually be manipulated in practice. The following papers in this series will link the potential nutritional impact to experimental evidence in horses as well as the practical implications of current knowledge on the prevention of developmental diseases in growing horses.

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## Appendix 1. Abbreviations and Glossary

|       |                                         |                                                                                                                                                                                                                                                                                                                                                        |
|-------|-----------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
|       | Annexin V                               | ion channel in MVs mediates the influx of $\text{Ca}^{2+}\text{Pi}$ from chondrocytes into MVs from Chondrocytes and from Matrix – binding sites can be inhibited by Mg, Zn, Cd                                                                                                                                                                        |
| ALS   | Acid Labile Subunit                     | produced in the liver by an ALS- gene, ALS is responsible for maturation of the Growth Hormones in the embryo                                                                                                                                                                                                                                          |
| ATP   | Adeno-tri-phosphate                     | Energy ‘currency’, releases energy for direct use when catabolysed to ADP (Adeno-di-phosphate)                                                                                                                                                                                                                                                         |
|       | Acidic Phosphatases                     | bind organic Phosphate                                                                                                                                                                                                                                                                                                                                 |
|       | Alkaline Phosphatase                    | liberates inorganic Phosphate from ACP inside MV - to form HAP inside                                                                                                                                                                                                                                                                                  |
|       | Calcitonin                              | systemic regulator hormone, acts on osteoclasts to inhibit their activity                                                                                                                                                                                                                                                                              |
| CCV   | Cartilage Canal Vessels                 | supply nutrients, minerals to subchondral zones and matrix from epiphysis, also to chondrocytes from growth plate / metaphyses                                                                                                                                                                                                                         |
|       | CAPPILLARY BUDS                         | well established in foetus, by 7mths no CCVs seen at any site (Carlson) end of CCVs reaching into lower hypertrophic zones/matrix or at epiphysis                                                                                                                                                                                                      |
|       | COLLAGEN TYPE I                         | mineralised by hydroxyapatite crystals - bone                                                                                                                                                                                                                                                                                                          |
|       | COLLAGEN TYPE II                        | part of soft cartilage, fairly durable, elastic                                                                                                                                                                                                                                                                                                        |
| ECM   | Extra Cellular Matrix                   |                                                                                                                                                                                                                                                                                                                                                        |
| DCAB  | Dietary Cation Anion Base in mEqu/kg DM | $(\text{Na}+\text{K}+\text{Ca}+\text{Mg}) - (\text{Cl} + \text{P} + \text{S})$ or $(\text{Na} + \text{K}) - (\text{Cl})$                                                                                                                                                                                                                               |
| GH    | Growth Hormones                         |                                                                                                                                                                                                                                                                                                                                                        |
| GAG   | Glycosaminoglycan                       | proteoglycan (PG) particularly important as Chondroitin-sulphate GAG chains                                                                                                                                                                                                                                                                            |
| HAP   | Hydroxyapatite                          | Mineralised $\text{Ca}^{2+}$ and P crystals TO FORM TYPE I COLLAGEN, first form inside MVs (intralumenal crystals) - grow until they rupture the MVs and released into Matrix where they bind to Type II Collagen and Type X septa in hyaluronic acid of matrix, HAP are integrated with Type II, X, PGs, degrading these Collagens to Type I Collagen |
| IGF   | Insulin Growth Factors                  | hormone regulating growth – also called somatomedins                                                                                                                                                                                                                                                                                                   |
| IGF-1 | Insulin Growth Hormone 1                | : released by liver and chondrocytes, stimulates chondrocytic IGF-2                                                                                                                                                                                                                                                                                    |
| IGF-2 | Insulin Growth Hormone 2                | : regulatory hormone during embryonic development, stimulates bone formation                                                                                                                                                                                                                                                                           |
|       | Lysil Oxidase                           | Pituitary Enzyme - inhibited by Corticosteroids. important in degradation of Type II Collagen and forming of Type I (bone) Cu as co-factor - can be inhibited by Zn, Mg, Cd                                                                                                                                                                            |



|                |                            |                                                                                                                                                                                                                                                                                                                                                          |
|----------------|----------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| LW             | Live Weight                |                                                                                                                                                                                                                                                                                                                                                          |
| mEq            | molar equivalent           | e.g. mEq of 3g Na = 3 x atomic/molar weight of Na = 3 x 22                                                                                                                                                                                                                                                                                               |
| MMPs<br>ECM    | Metalloproteinases         | Enzymes derived from chondrocytes , which produce and regulate                                                                                                                                                                                                                                                                                           |
| MV             | Matrix Vesicles            | released by Chondrocytes in the HZ, lipid membranes,                                                                                                                                                                                                                                                                                                     |
| OC             | Osteochondosis             |                                                                                                                                                                                                                                                                                                                                                          |
| OC             | Osteochondrosis Dissecans  |                                                                                                                                                                                                                                                                                                                                                          |
| Ob             | OSTEOBLASTS                | in subchondral Metaphysis, settle on calcified Septa/Trabecula to mineralise and synthesise woven bone (Type I Collagen), later in mature bone, secrete cellular factors, which regulate proliferation and differentiation of Oc                                                                                                                         |
| Oc             | OSTEOCLASTS                | in mature bone, to reabsorb Ca etc. from bone, break down structure to put stronger/denser matrix in place (by Oc) – especially during training/fittening , inhibited by Calcitonin                                                                                                                                                                      |
| PTH            | Parathyroid Hormone        | involved in ossification process, stimulated if Ca Serum low - leads to conversion in Kidney of 25-hydroxycholecalciferol into 1.25 dihydroxycholecalciferol, which stimulates Ca uptake in the gut via a receptor mediated mechanism - can cause Phosphaturia                                                                                           |
|                | Phosphatyl Serine          | from Chondrocytes forms Annexing V, = Acidic Phophatase                                                                                                                                                                                                                                                                                                  |
| PG             | Proteoglycans              | regulatory role; Inhibit excess mineralisation to keep external matrix 'soft' cartilaginous in hypertrophic zone, become shortened and more concentrated in lower hypertrophic zone - concentrated in mineralisation sites; allow/enhance mineralisation to take place in subchondral area                                                               |
| TGF            | Transforming Growth Factor |                                                                                                                                                                                                                                                                                                                                                          |
| TIMP           | Tissue Inhibitors          | of MMPs, inactivate and activate MMP's                                                                                                                                                                                                                                                                                                                   |
| T <sub>4</sub> | Thyroxine                  | increase rate of bone formation and resorption- important for penetrating cartilage cells, stimulated by excess carbohydrates (bone morphogenic proteins), Thyroxine (inactive) is converted to Triiodothyronine (active)<br>By iodothyronine 5' –deiodinase which is Se dependent and can be inhibited by excess Copper, but needs iron Fe - to convert |
| T <sub>3</sub> | Triiodothyronine           |                                                                                                                                                                                                                                                                                                                                                          |
|                | Zones                      |                                                                                                                                                                                                                                                                                                                                                          |
| RZ             | Resting Zone               | Zones along which chondrocytes develop during formation of bone tissue                                                                                                                                                                                                                                                                                   |
| PZ             | Proliferation Zone         |                                                                                                                                                                                                                                                                                                                                                          |
| MZ             | Maturation Zone            |                                                                                                                                                                                                                                                                                                                                                          |
| HZ             | Hypertrophic Zone          |                                                                                                                                                                                                                                                                                                                                                          |
| SCB            | Subchondral Bone           |                                                                                                                                                                                                                                                                                                                                                          |



# Effect of exercise and diet on the incidence of DOD

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## Introduction

The term developmental orthopaedic disease (DOD) was first coined in 1986 to encompass all orthopaedic problems seen in the growing horse and therefore encompasses all general growth disturbances of horses. It is non-specific and the definitions are not uniformly agreed. However, as defined by McIlwraith 2001, DOD may be taken to include Osteochondrosis (OC: 'a defect in endochondral ossification that can result in a number of different manifestations, depending on the site of the endochondral ossification defect' - one manifestation of which is osteochondritis dissecans (OCD) of cartilaginous origin); Subchondral cystic lesions (SBC); Physitis; Acquired Angular Limb deformities; Flexural deformities; Cuboidal bone malformation; Juvenile arthritis or juvenile degenerative joint disease and bony fragments of the palmar/plantar surface of the first phalanx of standardbred horses (believed traumatic lesions). It has been suggested that the clinical signs of OC occur only after a progression of events that begin with a disturbance in the normal development of the cartilage (sometimes referred to as dyschondroplasia; DCP) leading to OC. At this point physical stresses are superimposed, leading to clinical signs. It is also thought possible that the initial defects/ lesions may heal or develop into OCD or into SBC. Due to the multifactorial nature of DOD, no single cause is likely to result in expression. Factors that may contribute include a genetic disposition, biomechanical trauma, and mechanical stress through inappropriate exercise, obesity, rapid growth and inappropriate or imbalanced nutrition. Different combinations may be involved in different cases. Environmental or managemental factors most likely determine if expression occurs (i.e. provide the final triggering factor(s)).

Various nutrients have been highlighted over the years but in particular energy, calcium, phosphorus, copper and zinc and protein (Kronfeld, 1990; McIlwraith, 2001). Many studies have been undertaken and suggested to either support or refute the role of a particular nutrient. However, caution needs to be taken when evaluating and extrapolating from the various nutritional studies mentioned below. In particular it is important to remember that

- Energy is supplied to the horse via its diet but fundamentally energy is not a nutrient – and diets with the same overall energy content may have very different effects in the animal.
- Dietary energy is provided by the four principle dietary energy sources (hydrolysable carbohydrates, fermentable fibres, oils/fats, and protein). The relative proportions and characteristics of these sources within a particular diet may influence the physiological and biochemical responses to such a diet.
- Similarly not all protein sources are the same with respect to where they are digested and to what, as well as how the body responds to the products of digestion.
- Background differences in the climate, management practices, and pasture type and management may influence the response and therefore clinical outcome to a particular dietary manipulation, as can breed etc.

- Monitoring the influence of a particular nutrient without concurrently allowing for changes in the balance of other nutrients will complicate interpretation (e.g. copper and copper to zinc balance).
- Epidemiological studies in the field are very useful especially when they consider the make up of the nutrient supply and the balance rather than purely total intakes. Difficulties with adequate numbers and availability of reliable *individual* data can be major limitations for such studies.

## Energy and protein

### Energy

A number of studies have suggested that a high intake of energy may result in an increased incidence of DOD including:

- Feeding 129% NRC energy requirements to foals from 130 days of age resulted in an increased incidence of lesions compared with the control group (fed 100%) or those fed 126% of the National Research council's recommendations (NRC, 1989) for protein (Savage *et al.*, 1993a). Multiple lesions of dyschondroplasia (DCP) were found on gross post mortem in 11 foals fed the high DE diet; one fed the high protein diet and one fed the control diet. Histological lesions of DCP were found in 18 foals: in all 12 of the high DE, four of the high protein and 2 of the control foals.
- Cymbaluk *et al.*, 1990 reported ad libitum cube feeding resulted in a higher incidence of conformational and locomotor abnormalities at 25 months of age than the more restricted diet.
- Glade and Belling in 1984 compared the growth of foals fed either 70 (R) or 130% (H) of the NRC (1978) recommended levels for energy. The H group showed developmental disturbances of growth plates whereas the R group had normal development of the bones but at reduced speed.

However, it should be noted that there has been concern that the lesions produced by some of these studies are not directly comparable to those found in the field and many field studies have reported foals being fed much higher energy intakes without an apparent increase in clinical incidence (e.g. Kronfeld, 1990). This may be linked perhaps to the background level of predisposition within the individuals, nature of the energy being provided and the balance of the diet.

There has been a suggestion that if there are nutritionally induced patterns of acquired OC then this should be acknowledged rather than using the more general term DOD (Pool, 1993) for all. However, there are obvious practical limitations to this.

### Influence of feed type: role of the glycaemic response

Modern feeding and managemental practices have considerable influence on the health and welfare of horses (Davidson and Harris, 2002). Perhaps this is most noticeable in the growing horse, especially when under commercial conditions where the goal may be 'production' for early sale. In particular, this results in mares and foals either being kept on artificially enhanced pastures or being fed one to two large meals a day and the foals being weaned early (3-5 months compared with ~9 months in the wild). In such cases the diet typically consist of feedstuffs with a greatly reduced water content and often a radically different nutritional

profile from the diet that they would be able or would choose to select in the wild as a non-ruminant herbivore.

It has been suggested that diets which intrinsically produce high glycaemic peaks, or individual horses that respond to certain diets to produce, high glycaemic peaks (and subsequent effects on insulin and other hormones) may have an increased risk of developing DOD (Glade and Belling, 1986; Ralston, 1995 & 1996; Pagan 2001). Such diets have the potential to establish a feeding fasting cycle, which is a perturbation from hormonal patterns likely seen in grazing animals. This in turn may adversely influence bone development as the cyclical changes in glucose and or insulin may influence bone maturation via effects on the somatotrophic axis including:

- Growth hormone (Freud *et al.*, 1939).
- Thyroxine and Triiodothyronine (Glade and Reimers, 1985).
- Insulin-like Growth Factor I and equine chondrocytes (Cymbaluk and Smart, 1993; Henson *et al.*, 1997; Staniar *et al.*, 2001a; Staniar *et al.*, 2001b; Staniar *et al.*, 2002; Burk *et al.*, 2003).

Feeding diets, which provide the same energy intake, but which are derived primarily from oils and fibres rather than starch and sugars see Figure 1, may modulate these changes (Staniar *et al.*, 2001a; Hoffman, 2003).

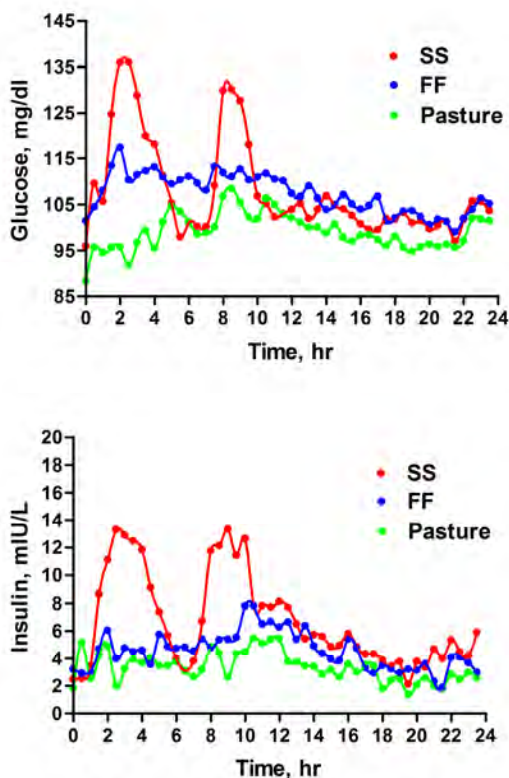


Figure 1. Glucose and insulin responses over 24hrs following the provision of two meals of feeds rich either in starch and sugar (SS) or fat and fibre (FF) compared with animals out at pasture with no supplemental feeds (Staniar *et al* unpublished data).

## Thyroid hormones and nutrition

Thyroid hormones are important in bone maturation and it has been suggested that the growth plates of the equine tarsal and carpal bones are particularly susceptible to the influence of thyroid hormones during the last months of gestation. Nutritionally too little or too much iodine in the diet may lead to goitre and various growth defects including inadequate ossification.

In humans it has been suggested that over-nutrition, regardless of its source will induce an increase in T3 production (Danforth *et al.*, 1979). High T3 subsequently inhibits TSH secretion and effectively lowers T4 concentration at the cellular level and may therefore affect bone development. However, in pigs the utilisation of thyroxine only increased when the energy intake increased due to the provision of additional glucose rather than via increased proteins or fats (Ingram and Evans, 1980). The same may also be true in the horse in that serum T3 levels have been shown to increase in direct proportion to the amount of sucrose given into the stomach – this was accompanied by a decrease in T4 levels after an initial increase (Biesik and Glade, 1986). Protein (casein) and fat, however, had no effect.

No studies to our knowledge have looked at thyroid hormones in detail and the incidence of DOD. However, this may be an area that is worth revisiting especially looking at how these hormones interact with the various other hormones and metabolites that have been highlighted more recently.

## Growth rates and energy intake

In other species, such as the pig, dog and sheep, evidence of increased osteochondral lesions, has been found in breeds with rapid growth rates compared with the slower growing breeds (Wolter, 1996; Goedebeure *et al.*, 1980; Nap, 1993; Scott *et al.*, 1996). A number of studies have linked rapid growth rates (and often by inference excessive feed/energy intake - see above) with an increased incidence of DOD.

- A detailed longitudinal growth study of 18 colts split into either limited or *ad libitum* fed groups illustrated more rapid growth and a higher incidence of clinically assessed conformational and musculoskeletal abnormalities in the *ad libitum* fed group (Cymbaluk *et al.*, 1990).
- Thoroughbred foals that developed cervical vertebral malformation tended to have higher weights and wither heights during particular periods of growth when compared to a group of normal foals (Ruff *et al.*, 1993).
- Osteochondrotic lesions were radiographically diagnosed in 27 Thoroughbred foals that also exhibited more rapid growth than 244 normal foals, between 3 to 8 months of age (Pagan and Jackson, 1996).
- A group of 15 Warmblood foals with osteochondrotic lesions in their stifle had significantly higher weight gains during particular periods of growth when compared to a group of 28 foals with no lesions (van Weeren *et al.*, 1999a).
- A survey of Standardbreds in Sweden suggested that foals which developed radiographic signs of tarsocrural joint OC had a higher BW at birth and continued to be heavier and have a significantly higher average daily weight gain than did the unaffected foals, (Sandgren *et al.*, 1993). This may indicate a genetic predisposition as these animals also had a larger frame than the horses without tarsocrural OC. Interestingly, no significant differences were found in weight or ADG in those affected or unaffected with Fetlock lesions. However, in contrast to a study by Wilke *et al.* (2003), there was a tendency for the horses that developed hock OC to be born later rather than earlier than the unaffected horses.

- Van Tilburg and Ellis (2002) correlated the growth rates of 144 Warmblood foals with radiological OC scores and found a significant increase in weight gain in the first 3 month period after weaning in OC positive foals in comparison to OC negative foals ( $p < 0.01$ ). There was no difference between groups in weights at birth in this study or in ADG outside the specified months.
- Borchers (2002) reports on an interdisciplinary research project to evaluate the development of body weight (BW) and withers height (WH) in Hanoverians with an additional aim of investigating the relationship of energy and protein intake on the incidence of OC. 83 different Hanovarian breeding farms in North Germany were visited monthly from March until October 2001. 687 foals born between Dec 2000 and July 2001 were weighed and measured monthly for a maximum of 6 occasions. Details of the feeding practices were recorded although similar to other epidemiological studies individual intakes are not known. The radiographic findings showed that 32% of the foals had evidence of OC (not necessarily associated with clinical signs). This study reported that, whilst colts were heavier than fillies from 4 months of age and month of birth had an effect on both BE and WH, there was no correlation between absolute body weight and withers height or average daily gain and growth rates of foals and the incidence of osteochondrosis. However, foals with fetlock OC lesions tended to be lighter than the OC negative foals. Foals with OC in the stifle however were heavier than the hock OC negative foals, suggesting that lesions in different joint locations may occur due to a different combination of factors.

These studies are used by some authors to suggest a connection of rapid growth with certain types of DOD. The connection, however, lacks strength because the chain of events between rapid growth and the molecular events that initiate DOD is a long one. Plus the multifactorial nature of this syndrome means that the connection is not always direct, for example in some animals a rapid growth rate could be a predisposing factor but expression might only occur when a number of other factors are superimposed (e.g. as a hypothetical example in those genetically predisposed to rapid growth, whose dams were fed inadequate copper during gestation and then the foals were fed an unbalanced diet as weanlings). Rapid growth may affect bone maturation directly or indirectly due to the mechanical overload or disturbances in nutritional supply and hormonal balance. These and other factors associated with rapid growth rates therefore may be involved to a greater or lesser extent in individual cases. It may be that the sometimes-inconsistent results found between studies are just a result of the nature of this condition.

Growth rate depends on both genetics and nutrition (Stanier *et al.*, 2004). Rapid growth rates and high-energy intakes are regularly included in the multiple causes of DOD. The direct relationship of these causes with the various manifestations of DOD has proven elusive. The difficulty is due to the complexities that relate energy intake to growth rate and normal or abnormal skeletal development. Especially when, in order for the rapid growth rate to occur a combination of genetic ability to grow rapidly and the nutrition to support at least in part such growth is required. Energy is available to the horse in numerous forms, each of which is used with a different efficiency and have distinctive effects on hormonal systems related to growth (somatotrophic axis). The relationship is further complicated by genetic differences. We suggest that to better resolve the role that growth rate and energy intake might play in DOD, both need to be investigated in much more detail.

It is also important to note that a lack of rapid growth does not necessarily protect against an increased incidence of DOD e.g. in Savage *et al.*'s 1993b study the high P diet resulted in lesions of DCP despite a trend towards a depression in average daily gain (non significant).

## Protein

Despite being linked in many horse breeders minds there have not been any conclusive studies showing adverse effects of high or low protein intakes on the incidence of DOD in the horse. In fact most studies have shown no effect of increasing protein on the incidence (Schryver *et al.*, 1987; Savage *et al.*, 1993a). It has been suggested (Ellis 2001) that the energy needed to remove ammonia from excess protein breakdown may in fact help to reduce the growth rate which may be supported by the studies of Hintz and Schryver (1972) and Thompson *et al.* (1998a & b). Recommendations with respect to total protein intake and incidence on DOD therefore can not be made.

However, it is appreciated in other species that particular amino acids are more prone to promote insulin secretion than others are but there has been little work done in this area in the horse. If diets that promote high insulin production are contraindicated for optimal growth then for the reasons mentioned above it might be advisable to avoid high amino acid intakes that promote insulin secretion (e.g. leucine and arginine). Other suggestions are that high protein diets may affect the dietary cation and anion difference (DCAD) resulting in increased urinary calcium losses due to an effect on the renal filtrate pH and subsequent decrease in calcium re-absorption (Frape, 1998). However, again there is no work directly linking this with the incidence of DOD and there is little evidence that this would play a role in the field (especially given that large changes in the DCAD are needed to affect renal pH in practice).

The recent study in Germany failed to find any apparent significant link between the feeding management of foals and the incidence of OC (Borchers, 2002; Wilke *et al.*, 2003). With respect to nutrition no relationship was found between the nutritional status of the mares (in relation to digestible crude protein and digestible energy) and the incidence of OC. However, for a number of reasons (e.g. when stabled, foals were not fed separately from the mares, estimations were used to determine milk and pasture intakes etc.) the nutritional status of the foals could not be determined accurately (Borchers, 2002).

Overall there is at the current time no scientific evidence for protein playing a major role in DOD in the horse.

## Intake and exercise

In a study of Dutch Warmbloods, Bruin and Creemers (1994) compared the incidence of OC in foals fed either high intake with high exercise (HH), high intake low exercise (HL), low intake high exercise (LH) and low intake low exercise (LL). They found that the HL and LH groups had significantly higher incidence of OC than the HH or LL groups with the highest incidence being found in the HL group. Suggesting again that it is likely to be a combination of factors that influence incidence and that neither high activity nor high intakes will always result in an increased incidence. The incidence in the HL group did come closest to the percentage of foals reported with OC in Warmblood horses in the Netherlands (Ellis, 2001) and in Germany (Wilke *et al.*, 2003). However, the lowest incidence was in fact seen in the HH group suggesting that if matched by intake exercise may be of value in reducing the incidence of OC.

In a follow up experiment on osteochondrosis and exercise (Van Weeren *et al.* (1999), foals were either kept in boxes and exercised (exercise comprised of a maximum of 3 minutes canter – in 30 second bouts - every 2<sup>nd</sup> day up to 5 months of age; n=14), boxed and not exercised (n=14) or kept in the field (n=15). It was reported that the growth rates of boxed



foals increased significantly compared to pasture groups during the third month and again in the fifth month of the experiment. This project did not aim to look at nutritional influences and kept all foals on equal diets of *ad libitum* grass intake. However, boxed foals received pre-cut grass, which could suggest that for a variety of reasons, in particular 'boredom' and the ready availability of grass, intake might have increased with a concomitant reduction in energy expenditure leading to the greater growth rates. But it is not known why this would occur just in these months. Although not statistically significant there was a tendency towards more severe lesions in the box rested foals (Van Weeren and Barneveld, 1999b). Exercise was not necessarily all good as some other potentially negative effects, were reported in the forced-exercised group (Van den Hoogen *et al.*, 1999).

The interrelationship between exercise and feed intake in relation to DOD is far from clear at this point although there is an indication that there needs to be a balance between these two factors and extremes must be avoided.

## **Minerals and trace elements**

### **Calcium (Ca), phosphorus (P) and magnesium (Mg)**

In an epidemiological study by Knight *et al.* (1985) carried out in the USA on 19 breeding farms with 384 yearlings, the highest problem scores were associated with low or deficient intakes of Ca and P or extreme ratios between these. However, to date there does not appear to be any clear evidence of a link between excess Ca and DOD or marginal P intakes and DOD.

Savage *et al.* (1993b) fed 30 foals either a control diet (C: 12 foals: according to NRC 1989 recommendations) or a diet with ~ 4 times the recommended intake for Phosphorus (HP: 6 foals) or one with ~ three and a half times NRC recommended levels of calcium (HC: 6 foals) or a diet with ~ 3.5 times Ca and 129 % of the NRC level for digestible energy (HCE: 6 foals). Clinical and radiographic signs of DCP were seen in 7 foals (4 on HP; 3 on HCE). Suspicious joint lesions of DCP were seen at post mortem in 13 foals (1 C; 5 HP; 2HC & 5 HCE) and histologically confirmed lesions of DCP were found in multiple joints and growth plates in 15 foals (2C; 5HP; 2HC; 6HCE). The lesions were more numerous and severe in the foals' fed HP and HCE than in the foals fed the C or HC diets. The authors concluded that high levels of dietary Ca did not appear to induce lesions of DCP in foals fed ~100% NRC recommendations for DE but neither did it reduce the incidence in foals concurrently fed the high intake of energy.

This group also looked at (Savage *et al.*, 1993c) the effect of feeding (6 foals in each group) either Control (C), high protein (HP) at ~126% NRC; high DE (HE: ~129%) or high P (HP ~ 4x NRC) diets on the growth of weanling foals over 16 – 18 weeks. Bone biopsies were taken after week 7/8 and then at the end of the trial. No significant differences were found with the initial biopsy, but by the end of the trial cortical bone porosity was greater in the foals fed HP than in the other groups. Three foals fed HE had joint effusion from week 13 - two of these and one other had radiological lesions consistent with DCP. One C foal had phytitis but no other clinical signs were seen in the other C or HP foals. Macroscopic lesions of DCP were found at post mortem in one HP foal, 5 HE and 5 HP foals. Histological lesions were found in 5 of the 6 foals fed HP and all foals fed HE and in 4 of the 6 foals fed HP (although in these the changes were minor). The changes in the foals fed high P were consistent, according to the authors, with a mild form of nutritional secondary hyper-parathyroidism although no clinical signs were noted. In this study high intakes of P appeared to result in an increased

incidence of lesions but little work has been carried out on the effects of P on cartilage development. The amount of exercise given to the foals, in this study, was intentionally limited and this may have played a role. As discussed below in the Knight *et al.* (1985) study an increased incidence in the field was not associated with high P intakes *per se*.

No adverse effects of Mg have been reported in relation to the incidence of DOD but little direct work has in fact been done on this in the horse despite the fact that Mg may compete for Cu binding sites and excessive Mg may inhibit PTH secretion (Ellis, 2001).

## Copper and zinc

A copper containing enzyme - Lysyl oxidase is involved in the X-linking of protein chains in elastin and collagen of cartilage. Disruption of these X-links due to copper deficiency may result in biomechanically weakened cartilage and increase the risk of DOD (Hurtig *et al.*, 1993). A number of studies have suggested a relationship between copper and zinc in the diet and DOD (Knight *et al.*, 1985; Bridges and Harris, 1988; see McIlwraith, 2001 and Table 1).

*Table 1. Some of the papers which have evaluated the role of copper in the incidence of OC in horses (from Ellis, 2001).*

| Author                                              |                      | Experimental copper levels<br>(g/kg DM Feed Intake)                                                                              | Daily Intake as<br>per experiment<br>or per estimated<br>feed intake* | Conclusions                                                                                                                                                                                           |
|-----------------------------------------------------|----------------------|----------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Cupps and<br>Howell, 1949                           | Foals                | 8-108mg/kg DM                                                                                                                    | *64 to 864 mg                                                         | 10mg/kg DM adequate                                                                                                                                                                                   |
| Knight et al.,<br>1990                              | Mares                | Concentrate feed: 13 or 32<br>mg/kg DM intake (last 3 – 6<br>months gestation and 3 months<br>lactation)                         | *132 or 356 mg                                                        | Reduced prevalence and<br>severity of OC with high<br>Cu inclusion.                                                                                                                                   |
|                                                     | Foals                | 15 or 55 mg/kg DM from ~ 60<br>days up to 180 days of age                                                                        | *55 or 201 mg                                                         | Time of feeding for foal<br>& mares unclear.                                                                                                                                                          |
| Hurtig et al.,<br>1993                              | Foals,<br>3mths +    | 8mg/kg DM<br>25mg/kg DM                                                                                                          | 48 mg<br>150 mg                                                       | 8 mg not adequate<br>rec.20-25mg Cu/kg DM                                                                                                                                                             |
| Pearce et al.,<br>1998a-c                           | Mares,               | Pasture for all plus half the<br>pregnant mares supplemented<br>with 0.5mg/kg BW/day<br>equivalent                               | *51 or 291 mg                                                         | Supplementing mares<br>leads to increased Cu<br>liver status in foals.                                                                                                                                |
|                                                     | Foals to<br>5 months | Pasture/mares milk for all<br>Plus half the foals<br>supplemented:<br>from 21 days: +0.2 mg/kg BW<br>from 49 days: +0.5 mg/kg BW | Intake unknown                                                        | Mare supplementation<br>decreased physitis scores<br>at 150 days plus reduced<br>incidence of articular<br>cartilage lesions on PM.                                                                   |
| Knaap et al.,<br>2000<br>Van Weeren et<br>al., 2003 | Mares<br>and foals   | Predominantly on pasture short<br>barned period (hay)                                                                            | unknown –<br>estimated range:<br>9 to 15 mg/kg<br>DM                  | No correlation between<br>liver copper in mares/<br>foals and OC status.<br>Foals with high liver Cu<br>at birth, had a higher<br>'recovery rate' from early<br>lesions – improvement in<br>OC score. |

The main storage organ for copper in adult horses is the liver. Liver copper levels, however, vary with age, diet and the individual animal. During gestation the total copper content of the

foetus increases mainly due to storage in the liver and at birth much higher liver copper concentrations are found than in adult horses (Gee *et al.*, 2000). Foals are born with variable liver copper stores. Liver copper concentrations of less than 400mg/kg DM in new-born foals have been suggested to reflect inadequate absorption or intake of copper by the dam (Meyer and Tiegs, 1995) although this has been questioned (Gee *et al.*, 2000). It was previously thought that the liver levels decline by 3 – 4 months to the adult hepatic concentrations (Cymbaluk and Smart, 1993). However, a small study suggested more recently that in some foals there may be a much slower rate of decline and the authors referred to these as ‘accumulators’ (Gee *et al.*, 2000). Neonatal foals obtain Copper primarily from milk although mare’s milk is low in copper and does not appear to be responsive to copper supplementation. Anderson (1992) reports an average of 0.2 mg/Cu/l milk and Pearce *et al.* (1998a) found levels of 0.32-1.7 mg Cu/l at birth, decreasing to a mean of 0.2mg/l after 14 days and remaining constant, thereafter. When doubling Copper and Zinc levels in the mare’s diet no effect on milk Cu and Zn were seen (Baucus *et al.*, 1987). Pearce *et al.* (1998a) also found no effect of additional Cu feeding pre-partum or post-partum on post-partum Cu concentration in the milk. Deficits in the suckling foals copper needs have been suggested to be met by mobilising hepatic copper (Cymbaluk and Smart, 1993) – which may explain the suggested value of copper supplementation in gestation (see below). Liver concentrations are thought to be responsive to dietary copper intake at least in the adult although the neonatal liver has a low protein synthetic capacity, which limits ceruloplasmin production. Plasma copper and CP concentrations are therefore low at birth but plasma Cu levels approach adult levels by around a month of age (Cymbaluk and Smart, 1993).

Knight *et al.* (1985) reported that the farms with the fewest problems fed rations that contained at least 2 times the levels of Ca, P and Zn and three to four times the level of copper then currently recommended by the NRC. However, similar to other epidemiological studies, there are a number of potential issues that need to be considered when evaluating the findings. Firstly it looked at the nutrition for the yearlings at this one point of time, rather than what the animals had been fed over their first year of life and it is not possible to determine accurately any relationship between the yearling diet and any creep feed. Secondly the farms with the fewest problems also kept the yearlings out of doors with access to shelter and water (it is therefore unknown whether these had more free exercise as young foals – see below). Despite these and other considerations the results are of interest especially given the large numbers involved. Hurtig *et al.* in 1993 reported that whilst all nine foals fed a diet containing 8 mg copper/kg DM intake developed OC lesions on post-mortem, only 5 of the 9 developed clinically significant lesions (and that therefore other factors influenced clinical expression). None of the foals in the group fed 25mg/kg DM developed clinically significant lesions (and in fact in two foals that joined the study at 3 months of age with mild clinical signs suggestive of DOD – these resolved during the trial) . In this study 120% of NRC energy and protein requirements were fed to both groups, potentially supporting increased growth.

Knight *et al.* (1990) looked at post-mortem cartilage changes in foals at 90 and 180 days of age. Mares were fed either 13 or 32mg/kg DM copper during the last 3 – 6 months of gestation and first 3 months of lactation and the foals were given creep feed with either 15 or 55mg/kgDM from an undisclosed time point but close to the 90 day cut-off. Foals remained in the experimental group to which their dam was assigned. Differences were seen in those euthanised at 90 days but the main differences appeared to be at the 180-day stage. Foals from the low copper group had more articular and physeal cartilage abnormalities at 180 days than those from the supplemented group. Lesions of OC were only seen in the unsupplemented

group. It is important to note that in this study it was not possible to determine the relative influences of the pre-partum and post-partum feeding.

Fujikawa *et al.* (1993) in their survey of 278 foals suggested that the highest incidence of epiphysitis of the fetlock joint in foals occurred on pastures with the lowest copper contents (< 6mg/kg) and there was no apparent relationship with calcium and zinc. Unfortunately it is not possible to determine what if any supplemental copper or other nutrients were provided in each case.

In a study in New Zealand (see Pearce *et al.*, 1998a-c ) copper supplementation of mares (n=12) in late gestation, with 0.5mg Cu/kg BW daily (~ 28mg/kg DM intake), significantly decreased the radiographic indices for physisitis in the distal third metatarsal bone of their foals at 150 days and the prevalence of articular cartilage lesions in comparison with the control group (n=9). Oral supplementation of the foals (with 0.2mg/kg BW daily from 3 weeks of age increasing to 0.5mg/kg daily) however, did not affect bone or cartilage compared with untreated cohorts. Supplementing the mares did increase the liver copper status of foals at birth significantly but had no effect on other tissue copper concentrations and there was no direct relationship between the radiographic physisitis scores and the foal liver copper contents. It is important to note that unlike many of the US and European based studies, articular cartilage lesions were minor in all foals with no evidence of clinical DOD *in vivo* with the exception of minor radiographic changes assessed at post-mortem. The differences in the results may reflect other nutritional, managemental and genetic factors. Copper supplementation of the foal did not have any effect on any of the bone or cartilage parameters but the copper intake of the control foals was only mildly deficient. In addition, the numbers were relatively low in each group. In this study the feed provided a low Calcium to Phosphorus intake and ratio (2.5-3.8g Ca : 2.3-4.3g P/kg DM) and there were very different copper to zinc ratios (6.2 & 1.7 : 1 for the unsupplemented and supplemented groups respectively).

A follow up study looked at the effect of copper supplementation by deep intramuscular injection during the ninth (100mg) and tenth months (250mg ) of gestation in mares pregnant to the same stallion (Gee *et al.*, 2000). In this small study copper injections had no effect on the liver concentrations of the foals at birth but this may reflect inappropriate dose, timing, method of administration etc. In the recent German epidemiological study, although accurate estimates of the foal's nutritional status could not be determined, it appears that there was a significant correlation between Cu intake and OC in the second period (3-6 months of age – P <0.05) with foals that were fed 10% below requirements showing greater OC (Granel 2002).

In a number of studies it has been suggested that early signs of DOD may resolve (Hurtig *et al.*, 1993; Van Weeren *et al.*, 1999) although there may be a time component to this. In fact it has been suggested that rather than low copper being a causative factor, an increased availability of copper may support various degrees of reparation. The studies reported by Granel (2002) and Pearce *et al.* (1998) could be taken as supporting this suggestion. A further study (Van Weeren *et al.*, 2003) correlated the liver copper status of mares and foals with OC scores – all animals had the same availability of feed but their estimated intakes of copper ranged from 95 – 153 mg/day and resulted in mean foal liver coppers of 372mg/kg at birth. No direct link between liver copper status of mares and foals at birth or radiographical OC development in foals at 5 or 11 months was established. However, foals with higher liver copper status at birth showed an increased recession of lesions before reaching the threshold age of 8 months. It could be suggested that a slightly better placental supply and greater liver

stores at birth helped in the repair process of lesions and that a good copper supply during gestation may be advantageous. This conclusion would be supported by the work of Pearce *et al.* (1998a) although further research is needed in this area on a much larger scale.

In conclusion, the more recent work confirms that copper supplementation may not be the 'magic bullet' suggested by some in the past as far as DOD is concerned. However, a reduced copper intake/absorption especially during gestation could possibly either be permissive to the development of DOD under certain conditions or may reduce the ability to repair lesions.

## **Zinc**

There have been several reports of industrially contaminated grass land with either zinc or cadmium resulting in an increased incidence of DOD (sometimes not confirmed histologically) in foals and youngstock (see Messer 1981; Bridges *et al.*, 1984; Eamens *et al.*, 1984; Campbell-Beggs *et al.*, 1994). Suggesting that increased exposure to zinc and possibly cadmium may result in the development of OCD. In the last report a severe case of OCD was reported in a 5month old foal raised on a drylot contaminated with mine tailings. High levels of zinc were found in the liver but there were 'normal' plasma and liver copper levels. Despite this the authors believed that the clinical signs were caused by zinc toxicity probably causing interference with copper. Interestingly the feed, water and soil all contained normal levels of zinc – milk contents were not evaluated however.

It has been reported that the typical gross articular lesions found in an earlier study of zinc toxicosis in horses grazing near a zinc smelter were not reproduced by feeding large doses of zinc (700mg/kg diet) to mares and mares nursing foals (Graham *et al.*, 1940). It has been questioned, however, whether the foals actually received the high zinc diet (Bridges and Moffitt, 1990). In a study to evaluate the influence of zinc on the ability of weanling foals to maintain normal copper balance (Bridges and Moffitt, 1990), ten foals of undisclosed previous history were weaned at 3 months of age and 4 were fed a control diet of ~8mg/kg copper and ~29mg Zinc per kg feed DM whereas 2 foals each were fed additional zinc oxide at either 250, 1000 or 2000mg of Zinc/kg feed DM. The foals fed the 2 higher zinc diets became hypocupremic within 5-6weeks and became lame due to cartilaginous disease, which the authors reported was similar to OCD with multiple lesions on histology. The other foals remained clinically normal.

Again there has been discussion regarding the nature and distribution of lesions caused by chronic zinc or zinc/cadmium toxicosis and whether such atypical patterns should be considered in the same way as the more *typical* DOD. It has been suggested that in fact they should be specifically referred to as 'zinc induced osteochondrosis' rather than being 'combined under the more general term of DOD' (Pool, 1993).

Zinc and cadmium may have an effect by competing with copper binding sites and the dietary ratio between copper and zinc may also be important (Spais *et al.*, 1977; Bridges *et al.*, 1984; Caure *et al.*, 1998). Zinc is believed to compete for the same transport mechanisms as Copper and is a potent inducer of intestinal hepatic and renal Metallothionein (MT) synthesis. It has been suggested that dietary Zn excess or high dietary Zn to Copper ratios may induce intestinal MT and form a non exchangeable Cu-MRT complexes in the GIT; alternatively absorbed Cu may bind with MT in the liver and kidney which may further aggravate a marginal or deficient intake (Cymbaluk and Smart, 1993; Campbell-Beggs *et al.*, 1994). The

dietary intake of zinc required to cause copper deficiency is likely to depend on the reserve amount of copper in the liver and other tissues.

An experiment in France attempted to assess Cu, by changing the diet on two studfarms (Caure *et al.*, 1998). The ratios of the elements were changed from Zn/Cu = 6.28 to Zn/Cu = 2.99 by increasing daily Cu intake from 93 to 211 mg. Results showed a significant reduction in OC scores on one of the farms (ratio 2.99) but the general design of the experiment did not allow for controlled conditions. Nevertheless, it highlights the importance of developing a correct ratio, which may be different between growing and adult animals.

The ratio of the elements in mare's milk is  $\sim \text{Zn/Cu} = 6.25$ . Pearce *et al.* (1998a-c) supplemented both Cu treatment and non-treatment mares with the same amounts of Zn, arriving at very different Zn/Cu ratios of 6.2 (no Cu supplement) and 1.7 (extra Cu) which may or may not have had an influence. According to requirement tables both treatment groups received the necessary zinc levels. Much more research is necessary to determine what the Zn/Cu ratio should be and what influence this may or may not have on the incidence of DOD.

## **Molybdenum**

Whether Molybdenum may interfere with copper in the horse has been disputed. In a relatively recent study (Pearce *et al.*, 1999) it was concluded that high Mb pastures (up to 15mg/kg DM) did not have an effect on copper status, at least in weanling fillies. Work in adult geldings would support this, one study having shown no apparent adverse effect of supplemental Mb up to 20mg/kg (as fed) for 21 days on the absorption and retention of Cu (Reiker *et al.*, 1999). Again to the authors' knowledge no scientifically reported increase in incidence of DOD with high Mb intakes has been reported.

## **Exercise**

It has been suggested that, unless severe lesions already exist, exercise may help to modulate the adverse effects of high-energy diets on joint development. As mentioned above Bruin & Creemers (1994) exercised warm-blood foals fed high energy diets (30-45mins of trot and gallop in a lunging ring several times a day) and found beneficial effects.

However, in a recent study exercise was not shown to reduce the number of lesions but it was suggested that it might help reduce the severity (Van Weeren and Barneveld, 1999b & c). OCD of the femeropatellar or tibiotarsal joint was examined in 43 foals of similar size all with reared to weaning at 5 months. 15 remained at pasture: 14 were kept in box stalls: 14 kept in same box stalls but were given an increasing number of gallops/sprints daily. 8 foals from each group were then euthanised at weaning whilst the remainder were given identical light exercise for an additional 6 months before euthanasia.

- Lesions were found in all foals at 5 months but the prevalence had decreased by 11 months
- Exercise did not influence the number of lesions but at 5 months there was a tendency for more severe lesions in the box rested foals (exercised and non-exercised).

The study concluded:

- Many lesions regress and do not become clinically apparent up to the age of one year
- Therefore the number of clinical cases may underestimate the total number of animals that have suffered

- The period during which a lesion develops and possibly regresses is limited and variable per site
- Exercise at the level given in this study may have some influence on the appearance and distribution of the lesions but does not appear to have an aetiological role.
- Exercise at pasture appears to be preferred.

In a 4yr study in Kentucky (Pagan and Jackson, 1996) it was hypothesised that in early born foals fetlock OCDs (as they defined them) could have resulted from inadequate sub-chondral bone formation. Due to restricted activity in foals born early in the year and housed indoors at night so that they were unable to withstand the forces put on the bone when they were allowed greater amounts of exercise as the weather improved. Hock and stifle lesions may have occurred in heavy foals that grew rapidly after weaning - increased intakes of high quality pasture may have contributed to this. In this study

- Fetlock OCDs tended to occur < 180 days of age
- Hock, shoulder and stifle OCDs occurred around 300 –350 days
- Those that developed hock and stifle OCDs as yearlings tended to be large foals at birth and grew rapidly from 3-8months
- Those that developed hock OCDs averaged 5kgs >Kentucky average at 25days and 14kgs heavier at 240days
- Those that developed stifle or shoulder lesions were ~ 5.5kgs > than the Kentucky average at 25days ; 17kg more at 120 and 12kg more at 300days
- Foals that developed fetlock OCDs before 6 months of age were born early in the year (Jan/feb/mar) but weight tended to be average.
- Foals that developed fetlock OCD at a later date tended to be normal size during the first 120 days but then grew heavier than average post weaning.

The recent large epidemiological study, carried out in Hanoverian Warmblood foals, suggested that time of birth had a significant effect on the incidence of OC (Wilke *et al.*, 2003) because it, in turn, was related to the amount of locomotion (free exercise) of the foals. When the researchers added together the OC findings in the fetlock and the hock the frequency of OC was 10% higher in the early born (before April) than in the late born foals (after April) which was statistically significant ( $P<0.01$ ). Within the fetlock joint alone the early born foals showed 6% higher incidence of OC than late born foals. The main seasonal effect was considered to be related to locomotion (free exercise) of the foals because feeding management was apparently ruled out as described above. Foals with very little locomotion (an average of less than five hours per day) within their first months of life showed the highest incidence of OC i.e. nearly 40% compared with 30% of foals which received an average of 13.5 hours per day and 25% in those that had an average of 20hours per day (Wilke *et al.*, 2003). Although exercise is obviously important it is not the whole answer given that even the high exercise group still had a high number of animals with the condition. It is also unclear whether the feed intake was adjusted for the foals according to their exercise level or not or whether there were differences in energy sources. Individual exercise patterns are not available.

Not all surveys have suggested, however, that exercise is beneficial. For example in the Sandgren *et al.*, 1993 study the incidence of hock OC in Standardbreds was higher in those born later in the year. These animals were, according to the authors, likely to have been given more pasture exercise than the early born foals – although the nature of the exercise was not accurately quantified.

It has been suggested that free pasture exercise is the best for the development of healthy cartilage resistant to injury (van den Hoogen *et al.*, 1999) and that conversely lack of exercise leads to a retardation of the normal development of the joint (van de Lest *et al.*, 2002). However, post-mortem examination of cartilage of the 'force-exercised' foals in the Van Weeren and Barneveld (1999) study showed an unresponsiveness to stimulation *in vitro* in comparison to the non-exercised and field groups, possibly making joints more receptive to ailments such as osteoarthritis (Van den Hoogen *et al.*, 1999). It may be that a certain amount and type of exercise may be needed to result in functional adaptation of the articular cartilage and that a key time for this process may be the first 4-5 months of age (Back *et al.*, 1999; Brama *et al.*, 2002; Wilke *et al.*, 2003).

In summary, it is currently recommended that with respect to exercise one should provide adequate exercise daily. However, more research is needed – in order to confirm, especially with respect to artificially (forced) applied exercise, how much, what type and when. Regardless, it may be advisable to avoid excessive strain or trauma to young growing bones and joints i.e. do not allow to exercise to fatigue/exhaustion but also avoid cooping up and then turning out abruptly especially on hard ground as this increases the risk of bio-mechanical trauma.

## **What about the future?**

The dynamic nature of growing animals enables them to adapt to an environment with characteristics that fall in certain ranges. The domesticated horse is subject to a range of environments today that in all likelihood is outside the range that it evolved for. We therefore need to move forward with studies that will better resolve how the horse's genetic characteristics interact with the environments that we provide as owners. It is likely therefore that obtaining a better understanding DOD and the role that nutrition and exercise (as well as genetics) play will require an integrative approach to be taken in future studies. Although the various studies outlined above do provide valuable insights and pointers into the potential importance of nutrition and exercise in this condition, in the future we may need to

- Differentiate the underlying cause (s) and the triggering factor(s) leading to any consequent clinical signs.
- Develop a better understanding of when, if at all, the critical developmental window for the initiation of any lesion may be affected by nutrition.
- Appreciate that the originating and triggering factors are likely to differ between the various conditions and between predilection sites within a breed.
- Appreciate more fully the differences as well as the similarities between lesions caused by our interventions and those that occur naturally and whether these have any practical relevance.
- Understand more the relevance of lesions found at certain ages and at certain sites either on post mortem or via imaging modalities on subsequent clinical effects and loss of performance.
- Understand the potential influence that nutrition may have on the ability of lesions to repair.
- Understand more clearly the interactions between diet (especially with respect to energy sources), hormones and growth.
- Use integrative physiology to better characterise the large 'black box' that currently lies between exercise, nutrition, plus genetic causes and the occurrence of DOD.



Other questions that, if answered, may help to increase our understanding of this complex syndrome include:-

- How influential (and what is being influenced) can relatively minor changes in nutrient intake really be – either positively or negatively?
- Is there a window of opportunity when such changes will have an influence either positively or negatively?
- Which nutrients are important – for what type of DOD and for which breed?

Finally in the future we may need to evaluate more than the ‘typical’ nutrient culprits or saviours – for example – can diet influence the activity of selenoenzyme iodine thyronine (important in the conversion of thyroxine into T3) and subsequently bone maturation; how important could Vitamin K be (important in osteocalcin production) especially in animals on a poor forage intake or with poor hind gut. Will the various oral proteoglycans or their next ‘generation offspring’ have any protective effect?

In conclusion, it appears that whilst nutritional factors and exercise may play a role in this multifactorial syndrome they are not the only factors involved and should never be considered in isolation. Current knowledge gives us some indications of the potential impact, under certain circumstances, that these can have but the precise interactions between themselves or with other contributing factors such as genetics is far from clear.

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## Feeding practices and prevention of developmental diseases

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### Introduction

Not only does the horse have exceptional physical abilities, it has also a fragile Orthopaedic structure. In view of its fast development and its increasingly early training, the young foal pays the highest price when it comes to Developmental Orthopaedic Diseases (DOD), thus compromising its athletic career.

For many years, the relationship between food and DOD was analysed from the point of view of the lack or excess in a particular nutrient: DODs were principally considered as part of nutritional aetiology: osteoporosis, rickets, osteofibrosis, osteopetrosis (Wolter, 1988; Paragon 2000). They still exist but are now seen as anecdotic in the larger breeding context, since the care given to animals is very high and the errors underlying these affections are better prevented.

Henceforth, multifactor DODs predominate, constituting a vast pathological complex uniting various clinical signs (angular warping of limbs, tendon spasms, limping) and a number of lesions (first of which being osteochondrosis). Although the clinical approach to these DODs is not different from previous ones, the analysis on their origins is more complex and, unfortunately, leaves us with many possible hypotheses leading to prevention methods that are not very orthodox sometimes.

These very protein-based affections (joint expansion, angular warping of limbs, cartilaginous degeneration or osteochondrosis) affect about 10 to 25% of the bone radii of the most vigorous show horses made to grow at highest speeds. For the majority of them, these lesions lead to clinical problems.

Four sets of various factors create together the conditions leading to the occurrence of DODs : genetic, biochemical, zootechnical and nutritional factors.

Genetic factors may act directly or indirectly. The genitors' contribution to their offspring's having various forms of DOD (balance problems, cervico-vertebral instability, osteochondrosis) has been clearly demonstrated and leads one to assume a high, although variable, rate of inherited DODs (Schougaard *et al.*, 1990). Indirect genetic factors may also prove very determining by creating a predisposition to fast growth, high body weight and fine bones. However, these genetic factors should not be overestimated nor used to give a fatalistic explanation to such a complex problem, nor considered a refuge or alibi for a passive attitude.

Biomechanical factors play a determining role through the constraints they impose on the growth cartilage. Although a well-planned physical activity may guarantee a harmonious muscular and skeletal development, repeating even slight traumatism on growth zones greatly increases the occurrence and strength of DODs in young horses (Jeffcott, 1991). Exercise as such is not a problem. However, problems arise with the increase of brutal

demands put on the horse and with making the horse go, without any transition, from a period of rest to a period of consistent effort. Such demands put a lot of stress on growing bones (Lewis, 1995). For instance, it is especially harmful to impose a complete open air activity to a horse that has been confined in a box for weeks. The newly formed bones will then be unable to bear increasing mechanical constraints. Any time a horse is inactive for 2 or 3 weeks, its return to physical activity should be gradual, in order to prevent DOD risks. Additionally, it is worth remembering that traumatism on growing bones do not only increase with the intensity of exercise, they also depend on the relation between weight constraints and the bone surface bearing the weight. As the animal's weight increases while its frame becomes finer, the risk of DOD increases. Hence, the risk of DOD is very low when it comes to rural horses (a more massive bone structure) and ponies (lower weight).

The focus on zootechnical performance constitutes the third component of DOD risks. It is probably the first risk factor. When growth speed is doubled (1050 g vs 670 g GMQ), DOD risks are multiplied by 4 to 5 times (Lewis, 1995), particularly for the initial growth period (up to 3 months old), which affects negatively the milk production of the brood mare. The problem is even bigger when the foal is adopted by a mare that is of a higher grade than its natural mother. Rapid growth (and thus a high energy supply) coupled with overly early physical activity constitutes the highest risk (Savage city, by Jeffcott, 1991). The bigger the animal's frame and the faster it grows, regardless of whether growth is due to genetic factors or a compensatory growth following a period of food restriction, the higher the risk of DOD occurrence for the foal.

Nutritional factors are the trickiest to analyse, since there are many factors to consider and knowledge on provisions to consider is approximate.

High energy supply is the most common cause of DODs. This contrary effect is at the same time direct (through the growth speed it occasions) and indirect. Indeed, by optimising growth, and thus also the tissues' nutrient needs, the energy supply can cause all others nutrients that are necessary to bone structure to have a limiting effect. Thus, a P rate of 0.25%, enough to satisfy the demands of slow growth, will no longer be sufficient for fast growth (0.25% will then be indispensable). A foal may then have an DOD as a result of giving it an energy supplement with an equally minimal Ca, Cu or Zn ration, hence the drop in bone density and in bone ash rate, creating the risk of DOD occurrence. The preexisting lack of these nutrients was simply hidden by the low initial energy supply level (such reasoning may also be valid for a low protein ration that is corrected using only this parameter). Here, the food balance notion finds its full justification. All the nutrients that are necessary to growth must thus evolve in a coherent fashion to keep a harmonious growth.

Protein nutrition has often been considered an important factor in DOD risks. It is true that bones, because they are partly constituted by proteins (20% of the total raw tissue weight), are highly sensitive to Nitrogen supplies. Low supply can reduce the growth capacity both for the length and width of the bone. But the shortage level necessary to the occurrence of this lack on the bone level is such (a lower than 9% level in total Nitrogen rich matter in each ration, i.e. bad hay) that it becomes unavoidably associated with a generalised malnutrition, little compatible with a sensible breeding method. The effects of Nitrogen excess are sometimes considered a risk factor. This hypothesis (already refuted by Shryver *et al.*, 1987) now seems little plausible. A protein supply that is higher than the estimated needs of the animal does not increase its growth speed. The growth rhythm of bones and muscles is genetically predetermined and cannot be exceeded once the energy needs

required for this growth have been met. However, growth may be made slower by a lack in energy or protein. Hence, the responsibility of a protein supplement for the incidence of a foal's DOD can only be explained by the improvement of Nitrogen levels in a ration previously lacking in protein and having an energy excess, leading then to a bone growth increase, but for a feeding context low in minerals (Ca and P) which generates a lack in the mineralisation of the bone matrix thus elaborated.

Evidently, calcium and phosphate nutrition is a guarantee of bone growth quality, as these two elements are major components of bones. A lack of calcium and phosphate supply may obviously lead to the growth's slowing down, as well as to a loss in bone density and to risks of DOD (Knight *et al.*, 1985). An optimal ratio should be sought between these two elements (CA/P between 1.0 and 1.5 if possible) but may also vary quite a bit without any major risk (between 0.8 and 3 according to Lewis, 1995), foals being especially able to tolerate an excess of Ca. The same is not true for P excess.

It has recently become evident that the relation between the foal's food and DODs is connected to the supply in copper and zinc. These two trace elements are highly present in bone construction (the first plays a role in constituting the proteic framework and the collagen, and the second in forming the bone crystal). Clearly, an insufficient supply (particularly through the mother's milk) may lead to a risk of deficiency predisposing the animal to DODs. A question then arises as to the use of any extra supply. The work of Knight *et al.* (1985 and 1990) and of Gabel *et al.* (1987) seems to demonstrate the use of such an extra supply over the usual recommendations (30 ppl against 10 for Cu and 80 ppm against 40 for the Zn) for rations given to foals to prevent DODs. These results were confirmed by Burton and Hurtig (1991). However, the recent work of Pearce *et al.* (1998) calls for evaluating the importance of previous research. While the Cu supply given to the brood mare improves the foal's cupric status at birth, a Cu supplement given to the foal and the mother at the pasture will only increase the hepatic stock without modifying in any important way the bone content and the rate of circulation. Complementary research is necessary to confirm the protecting role this element plays against DODs. A complete study of the data collected during the zoo-technical and food survey conducted in Basse Normandie on 439 foals (Paragon *et al.*, 2000) and of the data drawn from a radiological follow-up concerning 246 yearlings from that group (Valette *et al.*, 2000) may give a few answers.

In conclusion, while the foal's food does contribute in a major way to the zoo-technical results observed in breeding, it can only partially explain the occurrence of disorders in the young animal's locomotive system. Breeding is a whole that cannot leave aside any factors contributing to the creation of an accomplished show horse. However, following simple feeding rules will most often reduce the incidence of DOD: limiting growth under the mother; limiting energy intake (not too much cereal) for sensible growth; limiting the animal's access to legume forages and using mostly grass hay; giving a mineral supply that is adapted to the forage's composition.

On the initiative of Professor R. Wolter, and with the support of the Conseil Régional de Basse-Normandie, a large survey has been carried out on the breeders in three relevant departments (Calvados, Manche and Orne) between 1997 and 1999, in order to better grasp the breeding environment (behaviour, rationing), to quantify the major growth features of products, to identify the risk factors, in foods, that may lead to Orthopaedic disorders, and to evaluate the foals' Orthopaedic status.

In the present study, the major rationing features of mothers at the end of gestation and during the suckling period as well as the rationing features of foals 6 to 10 months old have been reported.

## Materials and method

### Animals

During the 1997-1999 period, the zoo-technical and feeding environment of 28 stud farms in the region of Basse-Normandie was put under study. Eleven of the stud farms are in the Calvados department, 7 in that of the Manche and 10 in the Orne. In total, data concerning 286 mares and 439 foals of the three breeds – English thoroughbred, French trotter and French saddle horse – were collected.

### Recording data

**Rationing:** During the 1997-1999 period, each stud farm was regularly visited at the rate of 9.2 visits per farm. At each visit, the rations given to mares were controlled (1256 rations in 1997; 926 in 1998) as well as the products (1357 rations intended for foals in 1998 and 623 to yearlings still present). At each visit, the feed used and the quantity of feed given were recorded. Samples were taken in order to carry out a bromatological analysis and to determine nutritional supply. Analysis involved: Dry matter (DM), total nitrogen matter (TNM), raw cellulose (RC), mineral matter (MM), phosphorus (P), calcium (Ca), iron (Fe), Zinc (Zn) and copper (Cu). The data obtained allowed us to figure out the quantities of Ufc, feed MADc and the DM/Ufc, MADc/Ufc, Ca/P, Zn/Cu and Ca/Zn ratios.

Data on the rations given to mares and foals at the various physiological stages were collected in order to establish an average ration for each animal category, physiological stage, stud farm and year. These average rations were used to figure out the nutritional supplies for each animal (gestating mares between 8 and 11 months, lactating mares between 1 and 6 months, and foals between 7 and 8 months old). These supplies were compared (Paragon *et al.*, 2001) to usual recommendations (NRC, 1989; Wolter, 1994; Martin-Rosset, 1990) and a profile was created (positive if there is an excess and negative if there is a deficiency).

**Radiographic score:** The radiographic score of each yearling, which reflects the foal's Orthopaedic status, was determined through the protocol established by Denoix *et al.* (2000), and an average radiographic score (aRS) was established for each group.

For the original 246 foals, the individual radiographic score varied from 0 to 18 (Valette *et al.*, 2000), whereas the average radiographic score for the groups of 0 to 7.79 foals was kept for this study. The 14 farms were chosen according to their aRS and classified into 3 lesion categories (Table 1).

*Table 1. Definitions of lesion categories.*

| Category | ARS (average RS for each farm group) | Number of breeding groups | Clinical signs                  |
|----------|--------------------------------------|---------------------------|---------------------------------|
| 1        | 0-1                                  | 4                         | Healthy horses                  |
| 2        | 2-3                                  | 5                         | Partly affected horses          |
| 3        | 4-7                                  | 5                         | Highly affected and lame horses |



For the 14 breeding groups, all information concerning food rationing for mares and foals was available.

## Statistical analyses

The data were processed using Discriminating Factor Analysis (DFA). This procedure allows us to define, per physiological stage, the aRS categories as a function of all the food indicators recorded and to identify the most discriminating characteristics between lesion categories.

These analyses also allow us to study the correlations between food indicators, to discuss the differences between physiological stages and to follow the rationing evolution from one year to the next.

## Results

Tables 2, 3 and 4 contain the food supply for gestating and lactating mares, and for 6 to 10 month old foals, as well as nutritional needs depending on the mares' and the foals' physiological state, estimated using NRC and INRA tables. Significantly different values have been set in bold letters.

*Table 2. Food supply for gestating mares.*

|                | Requirements<br>(NRC. INRA) | Least<br>DOD | Typical<br>Deviation | Medium<br>DOD | Typical<br>Deviation | Highest<br>DOD | Typical<br>Deviation |
|----------------|-----------------------------|--------------|----------------------|---------------|----------------------|----------------|----------------------|
| DM (kg)        | 9.1                         | 10.1         | 1.7                  | 10.7          | 0.8                  | 10.2           | 1.2                  |
| <b>UFC</b>     | <b>6.3</b>                  | <b>7.2</b>   | 1.3                  | <b>7.6</b>    | 0.8                  | <b>11.3</b>    | 0.8                  |
| MADc (g)       | 440                         | 780          | 358                  | 660           | 134                  | 840            | 137                  |
| Ca (g)         | 39                          | 53           | 19                   | 51            | 20                   | 61             | 20                   |
| P (g)          | 25                          | 29           | 13                   | 32            | 7                    | 39             | 4                    |
| Cu (mg)        | 205                         | 100          | 23                   | 145           | 54                   | 175            | 51                   |
| <b>Zn (mg)</b> | <b>645</b>                  | <b>305</b>   | 137                  | <b>425</b>    | 161                  | <b>590</b>     | 74                   |
| Ca/P           | 1.6                         | 1.8          | 0.5                  | 1.6           | 0.4                  | 1.6            | 0.6                  |
| Zn/Cu          | 3.1                         | 2.9          | 0.9                  | 3.0           | 0.7                  | 4.5            | 1.9                  |
| <b>Ca/Zn</b>   | <b>60</b>                   | <b>215</b>   | 48                   | <b>140</b>    | 47                   | <b>110</b>     | 42                   |
| DM/UFC         | 1.5                         | 1.4          | 0.6                  | 1.3           | 0.2                  | 1.2            | 0.1                  |
| MADc/UFC       | 70                          | 110          | 32                   | 90            | 10                   | 115            | 21                   |

The mares' feeding needs during the last three months of gestation are well met as far as Nitrogen, Ca and P are concerned, but the mares receive too much energy. The need for trace elements (Cu and Zn) is often not met, and Ca/Zn ratios are too high.

The food energy, Ca and P needs of lactating mares are quite well covered and without much variation. However, this is not the case for Nitrogen supplies, which are excessive and almost always higher than all the mares' needs. On the contrary, the need for trace elements (Cu and Zn) are most often not well met, they are even deficient and variable (variation coefficient from 40 to 70%).

Table 3. Food supply for lactating mares.

|                 | Requirements<br>(NRC, INRA) | Least<br>DOD | Typical<br>Deviation | Medium<br>DOD | Typical<br>Deviation | Highest<br>DOD | Typical<br>Deviation |
|-----------------|-----------------------------|--------------|----------------------|---------------|----------------------|----------------|----------------------|
| DM (kg)         | 12.5                        | 12.4         | 2.1                  | 12.2          | 0.7                  | 13.0           | 0.8                  |
| UFC             | 8.9                         | 9.9          | 2.0                  | 9.2           | 0.5                  | 9.5            | 0.6                  |
| <b>MADc (g)</b> | <b>850</b>                  | <b>1675</b>  | 495                  | <b>1265</b>   | 224                  | <b>1160</b>    | 241                  |
| Ca (g)          | 56                          | 103          | 12                   | 76            | 22                   | 78             | 14                   |
| P (g)           | 44                          | 48           | 8                    | 38            | 6                    | 49             | 8                    |
| Cu (mg)         | 205                         | 125          | 28                   | 150           | 18                   | 160            | 70                   |
| <b>Zn (mg)</b>  | <b>645</b>                  | <b>320</b>   | 94                   | <b>400</b>    | 88                   | <b>515</b>     | 153                  |
| Ca/P            | 1.3                         | 2.1          | 0.3                  | 1.9           | 0.7                  | 1.6            | 0.4                  |
| Zn/Cu           | 3.1                         | 3.0          | 0.7                  | 3.3           | 0.3                  | 4.1            | 1.3                  |
| <b>Ca/Zn</b>    | <b>86</b>                   | <b>290</b>   | 77                   | <b>200</b>    | 107                  | <b>140</b>     | 43                   |
| DM/UFC          | 1.4                         | 1.4          | 0.1                  | 1.4           | 0.1                  | 1.4            | 0.1                  |
| <b>MADc/UFC</b> | <b>97</b>                   | <b>173</b>   | 26                   | <b>127</b>    | 28                   | <b>128</b>     | 26                   |

Table 4. Energy supply for foals after weaning, between 6 and 10 months.

|                 | Requirements<br>(NRC, INRA) | Least<br>DOD | Typical<br>Deviation | Medium<br>DOD | Typical<br>Deviation | Highest<br>DOD | Typical<br>Deviation |
|-----------------|-----------------------------|--------------|----------------------|---------------|----------------------|----------------|----------------------|
| DM (kg)         | 5.8                         | 5.8          | 0.6                  | 6.4           | 0.5                  | 6.5            | 0.5                  |
| UFC             | 5.4                         | 4.9          | 0.4                  | 5.3           | 0.1                  | 5.6            | 0.5                  |
| <b>MADc (g)</b> | <b>575</b>                  | <b>480</b>   | 90                   | <b>540</b>    | 13                   | <b>610</b>     | 58                   |
| Ca (g)          | 30                          | 55           | 4                    | 45            | 10                   | 40             | 13                   |
| P (g)           | 20                          | 30           | 4                    | 25            | 0.1                  | 25             | 5                    |
| Cu (mg)         | 95                          | 145          | 10                   | 85            | 7                    | 100            | 71                   |
| Zn (mg)         | 360                         | 400          | 76                   | 540           | 93                   | 560            | 156                  |
| <b>Ca/P</b>     | <b>1.8</b>                  | <b>2.0</b>   | 0.1                  | <b>2.0</b>    | 0.4                  | <b>1.5</b>     | 0.4                  |
| Zn/Cu           | 3.7                         | 4.3          | 1.5                  | 4.9           | 1.3                  | 4.4            | 1.1                  |
| Ca/Zn           | 90                          | 115          | 32                   | 125           | 3                    | 120            | 23                   |
| DM/UFC          | 1.1                         | 1.3          | 0.1                  | 1.2           | 0.1                  | 1.2            | 0.1                  |
| <b>MADc/UFC</b> | <b>105</b>                  | <b>105</b>   | 3                    | <b>115</b>    | 3                    | <b>120</b>     | 19                   |

The way the foals' feeding needs after weaning and up to the age of 10 months are met is generally satisfying. Energy needs are accurately met. Nitrogen supplies are limiting after weaning but afterwards become balanced. Calcium and phosphorus supplies are more substantial. Despite the high variation coefficients (between 25 and 55%), few animals find themselves in a low food supply situation. This is not the case when it comes to the copper and zinc supply, which are on average satisfactory but have a variation coefficient of 50 to 100% at 6 months and 60% between 7 and 10 months. During all of the observation period, the Ca/P ratio remains very close to the objective sought, but the supply of Ca compared to that of Zn tends to be excessive.

## Relation between food supplies and the average radiographic score of breeding groups

The discriminating factorial analysis allows us to graphically represent the closest relations between food supply and the 3 lesion categories.

During the last three gestation months (Figure 1), a clear opposition appears between category 1, which includes the healthiest breeding groups, and category 3, which includes breeding groups with the most lesion affected or even lame foals. The latter category has the highest trace element content and excessive energy. The breeding groups in the three lesion categories are 100% classified in their original category, which means that all the breeding groups in a category are alike, not only as regards one but also all the twelve feeding variables observed.

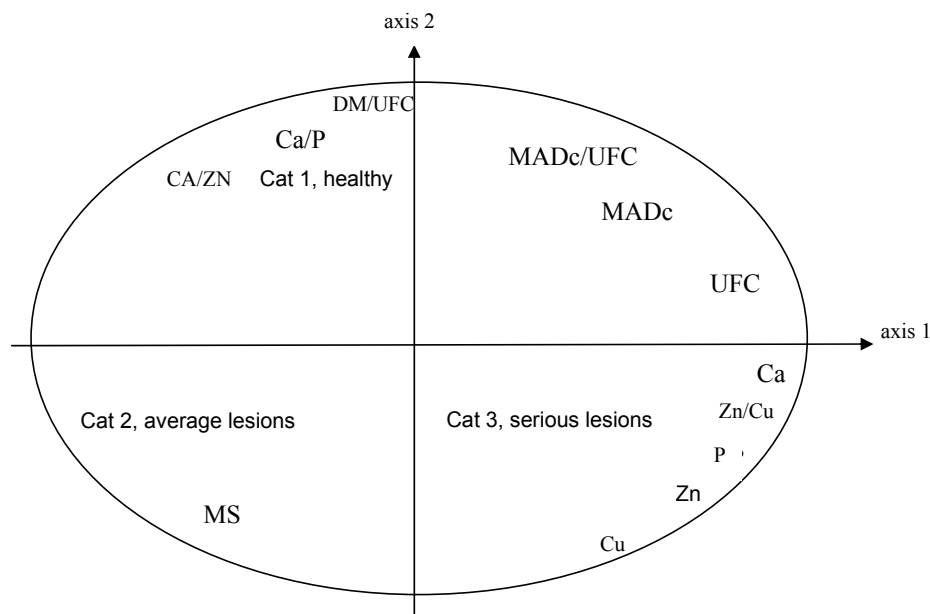


Figure 1. Layout of breeding groups in the AFD graphic representation of rations given to gestating mares.

During the 6 months constituting the lactating period (Figure 2), the opposition between Cat. 1 and Cat. 3 re-emerges. Category 1 shows the highest MADc, MADc/UFC and Ca/Zn values, the high Nitrogen content seems to play a protective role during lactation. As for the gestation period, the breeding groups from the three lesion categories are 100% classified in their original category.

During the foal's weaning to ten-month growth period (Figure 3), moderate Nitrogen and energy content as well as a Ca/P content close to 2 seem to be positive factors for well-balanced growth. An opposition appears here as well between Cat. 1 and Cat. 3 and the three categories are classified at 93%, an "average" breeding group has the same characteristics as a healthy breeding group.

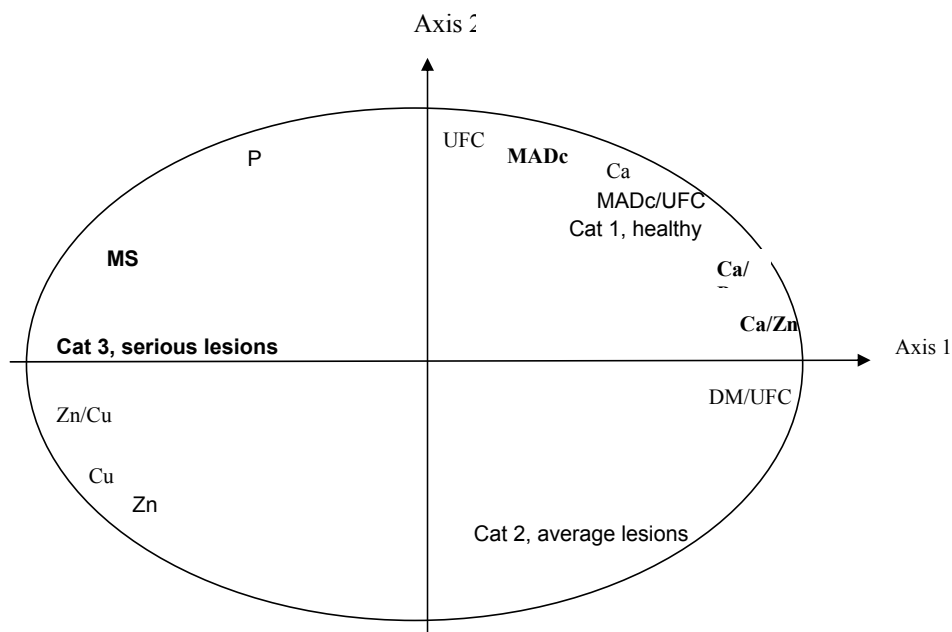


Figure 2. Layout of breeding groups in the AFD graphic representation of rations given to lactating mares.

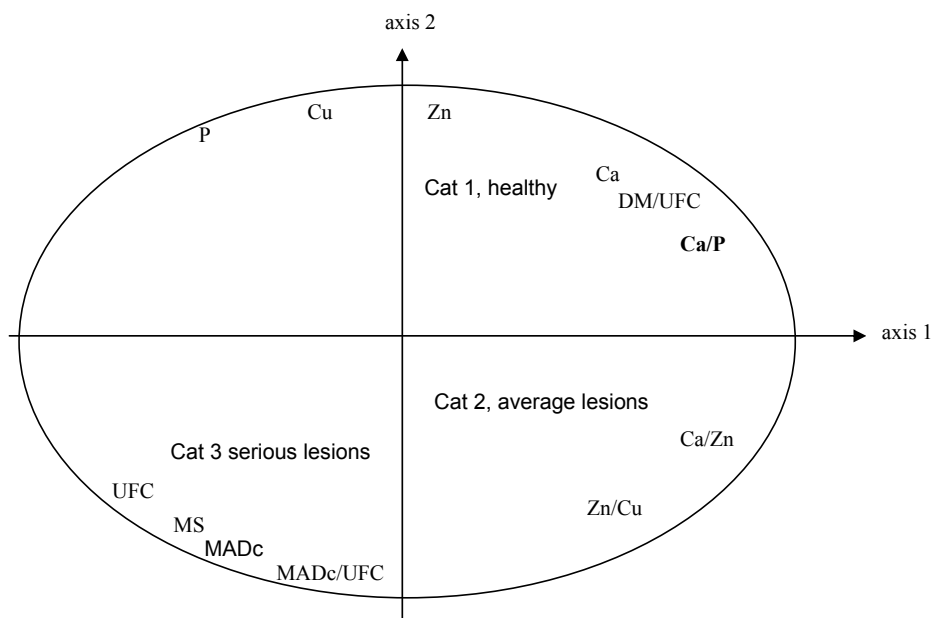


Figure 3. Layout of breeding groups in the AFD graphic representation of rations given to weaned foals.

On the whole, comparing these results to NRC and INRA values shows us that the breeding groups with the least lesions are those where gestating mares receive a supply of energy meeting their needs and a Ca/Zn ratio higher than 140, higher Nitrogen and calcium content, and a higher Ca/Zn ratio during the lactating period, and where their foals receive at weaning moderate nitrogen and energy supplies, a generous supply of Ca and a Ca/P ratio close to 25.

## Discussion

The nutrition hypothesis is only one of several others explaining the occurrence of foal DODJ. It is especially subtle to analyse, as many factors must be taken into account and the knowledge on supplies is often approximate, especially if we are trying, as is the case in this study, to consider not only the foal's food but its mother's as well.

An excessive energy supply is the most common source of foal DODJ (Jeffcott, 1991). Its effect is at first direct (through the excessive growth speed it causes). Such effect can be seen in our study, as the aRS is more positive in breeding groups where the energy supply given to foals 6 to 10 months old is most sensible. An indirect effect may also be described. Indeed, by optimising growth and thus the tissue's nutrient needs, the energy supply may cause all the other nutrients necessary for bone growth to have a limiting effect. The pre-existing shortage of these nutrients was simply masked by the low level of initial energy. The notion of food balance is hereby fully justified. All the nutrients necessary to growth must then evolve in a coherent fashion for a balanced growth to occur. This may explain the positive effect of having high levels of MADc and a high MADc/UFC ratio destined to lactating females on the aRS reported for the breeding groups of our study. Such supplies stimulate and optimise the mares' milk production without altering the milk's make-up, which will highly benefit the foal growing under its mother's care.

Protein nutrition plays a determining role in the foal's growth and the integrity of its bone and cartilaginous structure. While a supply shortage may reduce bone growth capacity as far as both length and width are concerned (Lewis, 1995), the shortage level necessary for this deficiency to become manifest on the bone level is such that it will become unavoidable associated with general malnutrition, little compatible with a sensible breeding environment. On the contrary, a reasonable supply seems to ensure bone growth with very low DODJ risks, especially after weaning. During this transitional period, moderate nitrogen supplies will limit the risk of indigestion and bad food absorption.

Calcium and phosphorus supplies will guarantee good quality bone growth, these two elements being major components in bones. A shortage of these elements leads to an obvious risk of slow growth, loss of bone density and more frequent DODJ (Knight *et al.*, 1985; Gabel *et al.*, 1987; Blanchard, 1994). In the present study, Ca and P supplies, meant for foals during their 6 – 10 month period, are generous as they come from Basse Normandie pastures (Doligez and Fouquet, 2000) which are high in Ca (on average 8 g/kg MS). A protective effect, against foal DOD, seems to be observed. This protective effect is made better through an excellent Ca/P ratio. According to Lewis (1995), the best Ca/P ratio is close to 1.5. Our study shows a significant protective effect for a Ca/P ratio close to 2, as the risk of foal DODJ increase when the supply decreases.

One of the recent findings regarding the relation between food and foal DOD involves copper and zinc supplies. These two trace elements are highly involved in bone construction. An insufficient supply (particularly through the mother's milk) may lead to a deficiency risk predisposing the animal to DODs. In our study, the lactating mares received poor supplies (10 to 12 ppm of Cu; 40 ppm of Zn), which copied the relative dearth of forage in the region (Doligez and Fouquet, 2000). On the other hand, the foals received a rich supply (16 to 20 mms of Cu; 70 to 80 ppm of Zn). However, no statistical relation could be established between these supplies and breeding groups' aRS. This leads one to question the use of supplements. The studies carried out by Knight *et al.* (1985 and 1990) and Gabel *et al.* (1987)

seem to demonstrate the use of such supplements beyond usual recommendations (30 ppm against 10 for Cu and 80 ppm against 40 for Zn) for rations meant for foals in preventing DODs. These results were confirmed by Burton and Hurtig (1991). However, the recent work of Pearce *et al.* (1998) leads to reconsider and limit the importance of previous studies. While Cu supply to the brood mare improves the newborn foal's cupric status (Pearce *et al.*, 1998a), a supply of Cu given to the foal and mother in the pasture will only increase the hepatic stock without significantly modifying bone content and the circulating rate (Pearce *et al.*, 1998b). Further research will be necessary to confirm the protective role of this element against DODs.

## Conclusion

The foal's food contributes in a major way to the zoo-technical results observed in the breeding environment but only partially explains the appearance of disorders in the young horse's locomotive system. Breeding is a whole that cannot neglect any factor contributing to the creation of an accomplished show horse. However, respecting a few simple feeding rules will most often limit the occurrence of DODs: limiting growth under the mother, limiting energy intake (not too much cereal) for sensible growth; limiting the access to legume forage and giving preference to mixed pastures; adopting a mineral profile corresponding to that of the forage used.

Horse breeding in Normandy, the country where this study was conducted, and where dietary errors are usually not encountered, is of high quality. For instance, a copper and/or zinc supplement, beyond the foal's requirements, did not constitute a significant preventing factor against DOD. This allows us to focus on specific nutrition aspects but also at the relationship between nutrition, breeding practices, genetics, DOD prevalence and intensity, and performance. More information is required to analyse more precisely the impact of the different dietary components, in relation to breeding practices. These studies, conducted in one of the world professional horse breeding farms, could lead, in the future, to determining the dietary requirements of race and show horses.

## Acknowledgements

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## Body composition in transition mares and suckling foals

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The aim of the present study was to test the hypothesis that body composition would change in transition mares and in early suckling foals during the first months of development. Eight Standardbred mares and their foals were used in this research. Mares weren't bred back after parturition. All mares and foals were fed with concentrate twice daily in individual stalls. They were fed pellets (15% CP and 3.00 Mcal/Kg DM) with amounts adjusted for stage of pregnancy and lactation to exceed in 10% NRC (1989) recommendations. Hay and salt were provided ad libitum. Biometric evaluations were made weekly starting two months before parturition and finishing three months after parturition. Body weight (BW) was obtained using an electronic scale and determination of fat mass (FM) made using the ultrasound measurement of rump fat thickness (Westervelt *et al.*, 1976). Data were analyzed using ANOVA for repeated measures and post-hoc the Tukey test. The null hypothesis was rejected when  $p < 0.05$ . During pregnancy, the mares gained BW ( $p < 0.05$ ) and Fat-free mass (FFM) ( $p < 0.05$ ) reflecting an increase of foetal size. However, FM and % body fat did not change ( $p > 0.05$ ). During this phase mares received energy supplementation 10% over the NRC recommendations. However, FM did not increase, probably because during late pregnancy, foetal growth is more dependent fat fuel than in early pregnancy (Fowden *et al.*, 2000). Thus, the level of supplementation was sufficient to promote healthy foetal growth without significant increase in FM in the mares. After parturition, the demands of lactation resulted in changes in body composition in the mares including a reduction of the FM ( $p < 0.05$ ) and % fat ( $p < 0.05$ ) without a change ( $p > 0.05$ ) in BW and or FFM. Foals underwent substantial changes in body composition during their first 3 months old of life. During the initial suckling, growth was rapid and body composition changed rapidly and the alterations were significant ( $p < 0.05$ ) for all characteristics analyzed (BW, FFM, FM and % fat). In conclusion, we observed that during pregnancy, mares alter their body composition by gaining weight and FFM. Body composition changed rapidly in foals during early lactation with fat mass accumulating as early as 7 d after their birth

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## **Developmental diseases of 230 foals in Basse-Normandie**

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### **Introduction**

Juvenile Developmental Orthopaedic Diseases (JDOD) cover all congenital or acquired disorders interfering with bone development (Pool, 1993). They affect most horse breeds and their occurrence seems to be increasing (Jeffcott, 1996). Our objective in this work is to establish the current widespread presence of these disorders in a population of French foals.

### **Materials and methods**

As part of the ESOAP programme (Elevage, Statut Ostéo-Articulaire et Performance (i.e. Breeding, Orthopaedic Status and Performance)), The growth of 230 thoroughbred, French Trotter, and French Selle foals, born in 2002 and 2003 in 19 breeding farms in Basse-Normandie was closely monitored. A complete motion examination was carried out at weaning ( $171 \pm 43$  days). The foals were filmed walking, from the front, from behind and from the side. Any likely posture defects, be they along a frontal plane (bowleg / Knock-knee) or along a sagittal plane (contractures / distensions), were reported. The behaviour of each member was reported on a scale of 0 (normal behaviour) to 3 (severely abnormal).

Radiographic tests, made up of 10 points (profile of front and back digits, carpal surface, profile of hocks, profile of stifles) were carried out on each member. Three veterinarians read the radiographic files, and the radiographic images were indexed and classified according to a standard protocol (Denoix *et al.*, 2001).

### **Results and discussion**

Only 72 foals (31.3%) were totally free of radiographic lesions. The foals had an average of 1.7 lesions (s.d. 1.9). The orthopaedic status was good for 46.3%, average for 33.9% and mediocre for 19.8% of the horses. Thoroughbreds showed a better orthopaedic status than did French Trotters and French Selles.

#### **Osteochondritis dissecant (OCD)**

With 171 radiographic images, OCD is the most frequent affection. It is essentially observed in the posterior fetlock (36.2% of lesions), the stifle (29.2%) and the hock (19.3%), but also in all other sites, except the feet.

#### **Bone cysts under the cartilage**

With 58 radiographic images, bone cysts under the cartilage are the second most frequent affection. They principally occur in the carp (63.8%) but can also affect all other sites.

## **Epiphysitis**

Radiographic images of epiphysitis were observed in 44 foals (19.2%). They mainly involve the back pastern (25 lesions) and the front fetlock (22 lesions), then the front pastern (16 lesions) and the front fetlock (15 lesions) and finally the carp (3 lesions).

## **Angular deformity of members**

Level 2 and 3 posture abnormalities in the frontal plane were observed on 58 foals (25.2%). Twenty-six foals had knockknee on the thoracic member and 14 on the pelvic member, 17 foals had a bowlegged thoracic member, and 9 had a bowlegged pelvic member.

## **Member contractures**

Grade 2 and 3 posture abnormalities on the sagittal plane were observed on 22 foals (9.6%). Three foals (1.3%) had carp contractures and 10 foals (4.3%) had digit contractures (clubfoot or knuckled-over posture). Radiography was carried out on four of them, of which two showed vertebral deformities.

## **Cervical vertebrae deformities**

Nine foals (3.9%) showed clinical signs that could be associated with cervical vertebrae deformities (hind member partial palsy, motion discordance, ataxia...). Radiography was carried out on four of them, two of which showed images of vertebral deformity.

## **Conclusion**

This work offers the first synthesis of all developmental diseases affecting French foals. It may serve as a basis for a wider epidemiological study of the factors constituting a risk of juvenile orthopaedic disease (diet, growth, genetics, breeding methods...).

Since bone condition evolves quite a bit in the months following weaning (Nannarone *et al.*, 2002), foals are observed until the yearling sales, and radiography is carried out on them a second time between the 15<sup>th</sup> and 18<sup>th</sup> months. This work will allow researchers to confirm the widespread presence of JDOD at a later age, just before the horse is put to work.

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# Changes in bone markers and intact parathyroid hormone with regard to the incidence of osteochondrosis in growing Hanoverian Warmblood foals

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## Introduction

Osteochondrosis (OC) is a developmental orthopedic disease due to a failure in enchondral ossification in the cartilaginous skeleton in foals. The cause of OC is multifactorial, some suspected factors are nutrition, endocrinology, growth and body size, genetic predisposition, and exercise. However, in recent studies a high OC score was accompanied by a lower bone mineral density in Dutch Warmblood foals (Firth *et al.*, 1999), which might reflect disturbances in growth rates as one of the contributory factors in the expression of OC. Other data suggested that housing growing foals with limited access to exercise might decrease bone mineral density (Firth *et al.*, 1999; Hoekstra *et al.*, 1999). As bone markers are known to reflect bone metabolism and intact parathyroid plays an important role in calcium metabolism, the present study was designed to monitor changes in bone markers and intact parathyroid hormone in growing foals with special emphasis on age, sex, growth rates, and osteochondrosis.

## Material and methods

629 Hanoverian Warmblood foals born between December 26, 2000 and July 01, 2001 (308 males, 321 females) from 83 farms in Germany were included in this study. Over a period of six months (from birth until six months of age) foals were monitored monthly (body weight, withers height, feeding practice, and housing conditions). Additionally, blood samples were collected and osteocalcin (ON, marker of bone formation, ELISA), PICP (carboxyterminal propeptide of type I collagen, marker of bone formation, RIA), ICTP (carboxyterminal telopeptide of type I collagen, marker of bone resorption, RIA), intact parathyroid hormone (intact PTH, RIA), total Ca (atomic absorption spectrophotometry) and P (spectrophotometry) were analysed in a total of 284 foals. Information on osteochondrosis was obtained between the fifth and tenth month after birth using x-ray (fetlock joints, hocks, and stifles). All results are presented as means  $\pm$  SD.

## Results

Bone markers in plasma decreased with age ( $P < 0.05$ ), whereas intact PTH remained nearly constant during the first months of life (Table 1). Sex did not influence these results.

Furthermore, there were no differences in bone markers and intact PTH between healthy foals (N = 165) and those foals that developed osteochondrotic lesions (N = 119).

*Table 1. Bone markers and intact PTH in plasma during the first 200 days of life in Hanoverian Warmblood foals.*

| age (d)   | ICTP [ $\mu\text{g/L}$ ] |                | PICP [ $\mu\text{g/L}$ ] |                | ON [ $\mu\text{g/L}$ ] |                | PTH [ $\text{ng/L}$ ] |                |
|-----------|--------------------------|----------------|--------------------------|----------------|------------------------|----------------|-----------------------|----------------|
|           | mean                     | $\pm\text{SD}$ | mean                     | $\pm\text{SD}$ | mean                   | $\pm\text{SD}$ | mean                  | $\pm\text{SD}$ |
| 1 - 50    | 38.8 <sup>a</sup>        | 6.92           | 913 <sup>a</sup>         | 139            | 172 <sup>a</sup>       | 61.2           | 27.5 <sup>a</sup>     | 33.8           |
| 51 - 100  | 34.0 <sup>b</sup>        | 5.85           | 876 <sup>b</sup>         | 127            | 135 <sup>b</sup>       | 41.2           | 35.7 <sup>b</sup>     | 37.7           |
| 101 - 150 | 30.1 <sup>c</sup>        | 5.00           | 821 <sup>c</sup>         | 145            | 104 <sup>c</sup>       | 34.6           | 31.6 <sup>ab</sup>    | 30.6           |
| 151 - 200 | 29.0 <sup>c</sup>        | 5.00           | 812 <sup>c</sup>         | 127            | 91.6 <sup>d</sup>      | 28.6           | 37.9 <sup>b</sup>     | 35.2           |

means in the same column with different superscripts are different ( $P < 0.05$ )

The decrease in plasma bone markers was accompanied by an increase in body weight and withers height. Growth rates were negatively related to bone markers ( $P < 0.05$ ) without any differences between OC affected and healthy foals.

However, different developments in plasma bone markers were noticed between foals, born early in the year (before 31 March, N = 154) compared to those foals who were born after the 31 March (N = 130, Table 2) without any distinctions between healthy foals and foals with OC. Additionally, body weight to withers height ratio was higher in early born foals. Intact PTH did not reveal these differences.

*Table 2. Plasma bone markers in early and late born Hanoverian Warmblood foals during the first 200 days of life.*

| age (d)   | ICTP [ $\mu\text{g/L}$ ] |                        | PICP [ $\mu\text{g/L}$ ] |                   | ON [ $\mu\text{g/L}$ ] |                    |
|-----------|--------------------------|------------------------|--------------------------|-------------------|------------------------|--------------------|
|           | early born <sup>1</sup>  | late born <sup>2</sup> | early born               | late born         | early born             | late born          |
| 1 - 50    | 38.2 <sup>a</sup>        | 39.0 <sup>a</sup>      | 883 <sup>a</sup>         | 940 <sup>a</sup>  | 173 <sup>a</sup>       | 172 <sup>a</sup>   |
| 51 - 100  | 33.7 <sup>b</sup>        | 34.3 <sup>b</sup>      | 869 <sup>a</sup>         | 883 <sup>b</sup>  | 142 <sup>b*</sup>      | 127 <sup>b*</sup>  |
| 101 - 150 | 31.0 <sup>c*</sup>       | 29.2 <sup>c*</sup>     | 836 <sup>a*</sup>        | 801 <sup>c*</sup> | 116 <sup>c*</sup>      | 89.5 <sup>c*</sup> |
| 151 - 200 | 30.6 <sup>c*</sup>       | 25.7 <sup>d*</sup>     | 835 <sup>a*</sup>        | 766 <sup>c*</sup> | 98.8 <sup>d*</sup>     | 80.3 <sup>d*</sup> |

means in the same column with different superscripts are different ( $P < 0.05$ )

\*differences between early and late born foals

<sup>1</sup>early born: foals born before 31 March, <sup>2</sup>late born: foals born after 31 March

## Discussion

As expected, an age-related decrease was noticed in bone markers, whereas intact PTH concentrations remained constant during the first 200 days of life. The development in bone markers and intact PTH was similar for OC affected and healthy foals. Amazingly, the fall in bone markers was different regarding the date of birth. The varying trend in bone markers was related to the housing conditions. Early born foals were housed in stables in the first months of life, whereas the late born foals had free access to pasture immediately after birth. Housing foals predominantly in stalls during wintertime may impair normal bone development, compared to foals maintained on pasture immediately after birth. Although there was no clear link between the development of osteochondrosis and bone markers, other orthopedic diseases

are implicated in growth. As bone development is affected by housing conditions, the measurement of bone markers might be a useful tool to monitor bone metabolism in growing foals.

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# Comparing bone metabolic parameters for Ardennes foals and draught horses

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## Introduction

Bones constitute a living tissue containing amorphous Calcium phosphate, easily put into use, forming hydroxyapatite crystals and surrounded by a protein matrix. The formation and resorption of bone tissue are two connected processes which are in balance and vary depending on the horse's age. The bone metabolism's intensity may be determined through a study of biochemical elements, certain minerals, enzymes, proteins or vitamins. The purpose of this paper is to report on the concentrations or activities of some of these metabolites for Ardennes foals and draught horses.

## Materials and methods

A group of 15 Ardennes draught horses, including ten foals 9 to 11 months old and five adults 4 to 10 years old, were used for this study.

The horses were kept on a diet composed of cereals and hay. They also had access to a meadow, which served as a paddock. The adults received 2 to 5 kg of the food mix every day, whereas the foals received 1 kg per 100 kg of live weight. The two food concentrates were different. Blood samples were taken from these horses at the end of the afternoon. Various parameters related to Ca metabolism were determined for these plasmas. The total Ca was analysed using atomic absorption. Ionised Ca ( $\text{Ca}^{++}$ ) concentrations were determined using a specific AVL 988-3 Analyser electrode. P, the total alkaline phosphatase (TALP) and the hydroxyproline (HYP) were calorimetrically analysed using a Technicon Autoanalyser. Vitamin D<sub>3</sub> (25 OH-D) and ostéocalcine (OC) concentrations were measured using radioimmunoassay. The bone alkaline phosphatase (BALP) was determined using an immunoradiometric method.

## Results and discussion

Table 1 summarises the chemical structure of the rations given to the two types of horses. On average and depending on the build, adult horses received between 58 and 67 kg of Ca and between 23 and 28 g of P every day. The foals received 63 and 27 g of Ca and P respectively. All these numbers are higher than those recommended by the NRC (1989) or the INRA (1990).

Table 1. Chemical structure of the rations given to Ardennes adult draught horses and foals (content expressed in % in dry matter).

|          | Prot. | ADF  | EE  | Ca   | P    | Na   | Mg   |
|----------|-------|------|-----|------|------|------|------|
| Adult h. | 11.4  | 32.9 | 1.4 | 0.52 | 0.36 | 0.22 | 0.14 |
| foals    | 10.1  | 35.6 | 2.4 | 0.74 | 0.31 | 0.20 | 0.16 |

The average concentrations of the various metabolites are reported in Table 2. They are compared to data on healthy half-breeds (de Behr *et al.*, 2003). No significant differences in Ca and Ca<sup>++</sup> content, representing about 50 % of the total Ca content, were observed between foals and adult horses. Foals had a significantly higher P content than the adult horses (1.8 vs. 1.3 mmol/l; P<0.05). Foals showed significantly higher concentrations and activity (P<0.05) than the adults for HYP (1.73 vs. 0.92 mg/l), TALP (392 vs. 322 u/l), BALP (5.31 vs. 38.4 mg/ml) and OC (9.2 vs. 30.2 mg/ml). This evidence suggests that foals undergo a higher activity of bone modification.

Table 2. Average concentration of the various plasmatic metabolites in Ardennes adult draught horses and foals, compared to concentrations in half-breeds.

|               | Ca (mmol/l)   | Ca <sup>++</sup> (mmol/l) | P (mmol/l)    | 25 (OH) D (ng/ml) |
|---------------|---------------|---------------------------|---------------|-------------------|
| Ard -adult h. | 2.8 ± 0.11 a  | 1.5 ± 0.06 a              | 1.3 ± 0.08 a  | 0.40 ± 0.32 a     |
| Ard -foals    | 2.8 ± 0.07 a  | 1.4 ± 0.02 a              | 1.8 ± 0.11 b  | 0.46 ± 0.26 a     |
| Half-breeds   | 3.0 ± 0.19    | 1.5 ± 0.14                | 1.1 ± 0.22    | 1.23 ± 0.62       |
|               | HYP (mg/l)    | TALP (U/l)                | BALP (ng/ml)  | OC (ng/ml)        |
| Ard -adult h. | 0.92 ± 0.26 a | 322 ± 53.7 a              | 38.4 ± 7.59 a | 9.2 ± 5.65 a      |
| Ard - foals   | 1.73 ± 0.26 b | 392 ± 45.1 b              | 53.1 ± 5.41 b | 30.2 ± 5.17 b     |
| Half-breeds   | 0.97 ± 0.29   | 190 ± 40.7                | 24.8 ± 7.27   | 6.3 ± 3.52        |

When these data are compared to those on adult half-breeds, it seems that the Ardennes horses had a higher bone modification than the half-breeds, if we consider the higher TALP and BALP activity. These observations are contrary to those reported by Lepage *et al.* (1998), comparing Franche Montagne and Ardennes horses to Swiss and Belgian thoroughbreds.

## Conclusion

The foal-adult draught horse comparison reported in the present study agrees with published data. However, the differences observed between draught horses and half-breeds underline the importance of carrying out studies on bone metabolism so as to assign draught animals to appropriate new activities, such as ploughing.

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# The influence of mineral supplementation on skeleton formation in Lusitano foals

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## Introduction

Functionality and behavioural characteristics of Lusitano horse make possible his use in very demanding equestrian disciplines, for a long period of his productive life. Since progressive training and learning submit the skeleton to considerable strengths, it is required to provide all the conditions that promote a normal bone and cartilage development.

Besides genetic and exercise, nutrition has been implicated as a major factor for an adequate skeletal development in horses, mainly, on diet's mineral content.

Previous experimental research has shown that bone metabolism can be assessed by bone biochemical markers like bone alkaline phosphatase (BALP) or osteocalcin (BGP) (Lepage *et al.*, 1990, 1991, 1997 and 1998; Price *et al.*, 1995 and 2001; Black *et al.*, 1999; Toquet *et al.*, 2001).

The main objective of this study was to evaluate the effect of Ca, P and Mg supplementation of traditional diets, on bone formation in Lusitano breed foals, from weaning to one year old.

## Materials and methods

### Animals and data collection

Two groups of eight colts each (258-d average age), received for 140 days (between December 2001 and April 2002), iso-energetic and iso-proteic diets, that differed only on Ca, P and Mg levels (respectively, 0.26%, 0.27% and 0.12% vs. 0.56%, 0.43% and 0.18%).

The basal diet consisted of traditional feeds (oat, horse bean and oat hay) to provide INRA (1990) recommended nutrient allowances for moderate growth. Both groups (A and B) were supplemented with trace minerals and vitamins, but only group B was supplemented with Ca, P and Mg.

The animals (8+8) were housed in two 150 m<sup>2</sup> covered parks. All foals received a diet with no macro-mineral supplementation for a 15 day adaptation period.

Venous blood samples were collected periodically (0, 35, 70, 105 and 140 days) for the evaluation of plasmatic concentrations of Ca, P, Mg, Cu, Zn, parathormone (PTH intact molecule), bone alkaline phosphatase (BALP) and osteocalcin (BGP).

## Statistical analysis

Shapiro-Wilks and Kolmogorov-Smirnov tests were carried out. All variables had a normal distribution, except PTH and BALP. Ca, P, Mg, Cu, Zn and BGP concentrations were analyzed by variance procedures for a repeated measures design. Analyses were performed with SAS by GLM procedure. Pearson's correlation coefficients were used to examine the variables relationships. PTH and BALP data were analyzed by Mann-Whitney and Wilcoxon non-parametric tests.

## Results

Concerning minerals plasmatic concentrations, no overall significant differences were observed between both treatments ( $P>0.05$ ) except for P on days 70 and 140, Cu on day 70 and Zn on day 35 (Table 1).

*Table 1. Effect of mineral contents of the diets on Ca, P, Mg, Cu, Zn and Osteocalcin plasmatic concentrations.*

| Groups                          | Day of the study |    |            |    |             |    |             |    |             |    | Model          |        |        |
|---------------------------------|------------------|----|------------|----|-------------|----|-------------|----|-------------|----|----------------|--------|--------|
|                                 | 0                |    | 35         |    | 70          |    | 105         |    | 140         |    | R <sup>2</sup> | CV     | P      |
| Ca (mg/dL) (LSMean±SE)          |                  |    |            |    |             |    |             |    |             |    |                |        |        |
| A                               | 10.54±0.34       | ns | 9.75±0.34  | ns | 9.22±0.34   | ns | 10.65±0.34  | ns | 10.75±0.34  | ns | 0.499          | 9.30%  | 0.0044 |
| B                               | 10.46±0.34       |    | 9.91±0.34  |    | 9.29±0.37   |    | 10.88±0.34  |    | 10.63±0.34  |    |                |        |        |
| P (mg/dL) (LSMean±SE)           |                  |    |            |    |             |    |             |    |             |    |                |        |        |
| A                               | 6.46±0.23        | ns | 5.95±0.23  | ns | 5.16±0.23   | *  | 5.34±0.23   | ns | 6.71±0.23   | *  | 0.542          | 10.83% | 0.0008 |
| B                               | 6.88±0.23        |    | 5.46±0.23  |    | 5.92±0.25   |    | 5.52±0.23   |    | 6.02±0.23   |    |                |        |        |
| Mg (mg/dL) (LSMean±SE)          |                  |    |            |    |             |    |             |    |             |    |                |        |        |
| A                               | 1.33±0.06        | ns | 1.23±0.06  | ns | 1.37±0.06   | ns | 1.02±0.06   | ns | 1.07±0.06   | ns | 0.582          | 14.84% | 0.0001 |
| B                               | 1.32±0.06        |    | 1.12±0.06  |    | 1.34±0.07   |    | 0.96±0.06   |    | 0.98±0.06   |    |                |        |        |
| Copper (µg/dL) (LSMean±SE)      |                  |    |            |    |             |    |             |    |             |    |                |        |        |
| A                               | 120.71±5.27      | ns | 100±5.27   | ns | 138.05±5.27 | *  | 121.41±5.27 | ns | 107.91±5.27 | ns | 0.638          | 13.16% | 0.0001 |
| B                               | 124.91±5.27      |    | 99.56±5.27 |    | 116.26±5.72 |    | 107.72±5.27 |    | 98.99±5.27  |    |                |        |        |
| Zinc (µg/dL) (LSMean±SE)        |                  |    |            |    |             |    |             |    |             |    |                |        |        |
| A                               | 53.49±6.04       | ns | 47.91±4.02 | *  | 31.22±4.02  | ns | 27.09±4.02  | ns | 40.75±4.02  | ns | 0.531          | 30.72% | 0.0074 |
| B                               | 46.27±6.05       |    | 32.98±4.02 |    | 30.4±4.39   |    | 35.6±4.02   |    | 36.77±4.02  |    |                |        |        |
| Osteocalcin (ng/mL) (LSMean±SE) |                  |    |            |    |             |    |             |    |             |    |                |        |        |
| A                               | 52.25±3.44       | ns | 51.74±2.88 | *  | 61.05±2.88  | ns | 65.18±2.88  | ns | 79.17±3.14  | *  | 0.81           | 14.09% | 0.0001 |
| B                               | 43.98±3.12       |    | 42.06±2.88 |    | 52.66±3.12  |    | 59.57±2.88  |    | 70.24±2.88  |    |                |        |        |

From day 0 to the end of the study we observed an increase in average plasmatic concentrations of osteocalcin in both groups. Significant differences ( $P<0.05$ ) between treatments were observed on days 35 and 140. For this period, a positive correlation was observed with age (0.626;  $P<0.001$ ).

PTH plasmatic concentrations varied between 29.8±4.0 and 118.9±26.1 pg/mL on group A and between 30.6±7.4 and 95.3±33.0 pg/mL on group B. There were no significant differences between groups. However, PTH concentrations differed significantly ( $P<0.05$ ) with sampling period.

In respect to BALP plasmatic concentrations, significant differences ( $P<0.05$ ) were observed with sampling period, with an inverse correlation with age (-0.565,  $P<0.001$ ). Values varied

between  $229.6 \pm 39.4$  and  $96.8 \pm 5.3$  U/L on group A and between  $275 \pm 32.7$  and  $105.3 \pm 5.6$  U/L on group B. Significant differences ( $P < 0.05$ ) between groups only occurred on 35-d.

## Discussion

Plasmatic concentrations of the analyzed minerals are in agreement with those reported (Bauer *et al.*, 1984; Nielsen *et al.*, 1998; Olddruitenborgh-Oosterbaan *et al.*, 1999) for other horse light breeds.

BALP results confirm the inverse correlation between age and this bone marker, which has been already observed in longitudinal studies in Thoroughbred horses (Price *et al.*, 1995, 2001).

Age-related changes in plasma osteocalcin have been previously reported for horses (Lepage *et al.*, 1990, 1991, 1992; Bell *et al.*, 2001; Price *et al.*, 2001). However, the present data are in agreement with the transient increased levels observed by Price *et al.*, 2001, on the same period of the year and with similar aged foals. A possible seasonal effect on the osteoblastic activity could be suggested by the increase in the osteocalcin levels.

Overall, no significant differences were observed between both treatments. Since Ca, P and Mg levels in the control diet were close to supply the animal needs and the fact that the foals were confined, could explain the observed results. The evaluation of bone mineral density should be considered in future studies for a better understanding of bone markers in the horse.

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# Clinical use of naloxone/calcium association in the meconium impaction and prevention of stress-related disorders of weaning in foals

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## Introduction

First 24 hours of life and weaning of foals are two critical and stressful conditions in which gastrointestinal and skeletal pathologies, can arise. Neonatal meconium impaction show characteristic symptoms: restlessness, reduced frequency of nursing, nervous walking-box with elevated tail, swishing of the tail, colic pain, rolling, dorsal recumbency, straining with kyphosis, (Traub-Dargatz, 1992; Bucca, 1997; Bernard, 2002, Lacalandra *et al.*, 2001). Retained meconium, if not promptly diagnosed and treated, can induce serious consequences and death. For resolution, saline and paraffin enema was indicated or enterectomy if the impaction is located in the large colon. This pathology is principally linked to lack of colostrum assumption, but the functional cause is the reduced intestinal motility (faecal stasis), which is related to a reduced internal concentration of calcium ions in the smooth muscular cells. These cells are in connection with neuronal circuits in the gut wall, and those of non-neuronal “pacemaker” cells (Hirst, 1999). Binding studies have documented the presence of opioid receptors on nerve and smooth muscle in the gastrointestinal tract (Kummerle *et al.*, 1992; Bagnol *et al.*, 1995). The high level of endogenous opioids is capable of interacting with the balance between intracellular and extracellular calcium levels. Mainly in the periparturient period calcium turnover can be negatively influenced by the high level of  $\beta$ -endorphin in mother circulating blood and consequently at foetal/newborn circulation (Minoia and Sciorsci, 1994). Naloxone (antagonist of Endogenous Opioid Peptides) in Calcium Gluconate solution, removes endorphins from the specific cellular membrane receptors, re-establishing the membrane potential and functionality of smooth muscular cells (Sciorsci *et al.*, 2000a). In the colic syndrome of adult horses this pharmacological association showed its therapeutic efficacy (Sciorsci *et al.*, 2000b).

Also the weaning can be very stressful in the foal. Low attention to this transition time and the inadequate pre-weaning food preparation, the different methods to wean foals can determine: self injuries due to the panic behaviour (Waters *et al.*, 2002), reducing of immunity and resistance to diseases, less food assumption, improperly balanced feeding diets with digestive problems. In this situation an increase of cortisol (Malinowski *et al.*, 1990; Hoffmann *et al.*, 1995), and/or a transitory hypocalcemia (Arnbjerg, 1980), were observed. Moreover after weaning, as reported by Flether *et al.* (1998), there is a reduced bone metabolism (decline of osteocalcin), which can induce various pathologies such as osteochondrosis, epiphysitis, angular deviation and deformities.

In this work, the results of the therapeutic use of Naloxone/Calcium Solution in newborn foals with meconium impaction and as a stress preventive in the weaning of foals, are reported.

## Material and methods

Forty-five newborn foals with meconium impaction were treated from 18 and 30 hours after birth, in four years of clinical activity (1999-2002). Diagnosis was based on the anamnesis and clinical observations. In all subjects, 0.1 mg/10kg/bw Naloxone (Naloxone Hydrochloride, Diosynt, Holland) in 0.5 ml/10kg/bw 20% Calcium Gluconate (New ICC, Upjhon SpA, Italy) was intravenously administered. Treatment always included the use of warm water or paraffin solution (0.5 to 1 L), given gently per rectum with a soft flexible tube. Time from treatment and meconium expulsion, gut contractility and the principal body parameters as heart rate and respiratory function, were monitored.

In the 2002 year, at the same doses, the experimental therapy with Naloxone/Calcium solution, was daily injected in 12 weanling trotter foals for 7 days. A control group of 8 subjects in the same herd, was not treated. The weaning method we utilised, was: parasitic control and worming, grouping the foals by two or more of same sex and age ( $180 \pm 20$  days), separation from mother during the day-light in the first 3 days of treatment and then complete separation, free access in a paddock with a mixed grass (clover, purple medic) plus a pelleted concentrate specifically formulated for colts of 1-8 months in age. The recorded parameters in both groups, were: complete assumption of food, presence of anxiety, pawing, restlessness, vocalisation, frequency of locomotor activity, wood-chewing. Three months later (9-10 months of age), delayed growing general condition and normal plumb lines, was evaluated together with radiological exams of joint spaces.

## Results

In most of the foals with meconium impaction, a transitory increase of sensibility to abdominal pain with the typical signs of colic syndrome, was observed few minutes after administration of Naloxone/Calcium association. Subsequently, 25-30 minutes later, all foals urinated and became quieter, showing a marked reduction of tachicardia and sweating. Respiratory function became normal and auscultation of abdomen revealed a progressive increase of gut motility until complete evacuation and recovery. In some cases, on the basis of the results of the first administration and of the general conditions, therapy was repeated 30 minutes later. Only few cases needed Ringer's Solution infusion.

The parameters observed in the weanling foals treated with Naloxone/Calcium indicate the improved welfare of foals during weaning. They showed a good appetite with complete assumption of food, lack or strongly reduced panic behaviours, calm at pasture and easy handling, growing was regular. No radiological joint pathology was recorded at 9-10 months. Only one foal revealed the appearance of splints. In the control group on the contrary a partial assumption of food was observed together with an elevated frequency of vocalisation (whinnies) and nervous walking at pasture. These manifestations continued for about 12-15 days inducing, in some subjects, a clear weight loss. Three foals of the control group showed plumb lines defects, splints and in one foal, at radiological control, presence of osteochondrosis.

## Conclusions

Uterine contractions and placental detachment provokes the passage of EOP in the foeto-maternal circulation which increase EOPs in the newborn foals. Binding of free opioids at their specific gut receptors may alter intestinal motility. The positive results obtained by



utilising antagonist of EOP receptors (Naloxone) and Calcium Gluconate in the meconium impaction of foals, support the hypothesis that the high endogenous opioidergic tone is involved in the pathogenesis of the irregular of gut contractility. Naloxone/Calcium association is an innovative therapy and it might be also considered as a prophylactic treatment on all newborn foals.

A new grouping of foals, the separation from the mother and the food variation are recognized causes of stress in young horses at weaning. The increase of EOPs alter bone metabolism and may inhibit pre-synaptic release of acetylcholine with negative influence on the neuromuscular end plates activity. The consequent susceptibility of limbs to injuries and pathologies in this period suggest the opportunity of prevention of stress at weaning.

The obtained data indicate the possibility of a systematic use of Naloxone/Calcium before and during weaning in foals. Moreover, the complexity of factors involved in the weaning stress-pathologies of horses need further and more extensive studies supported by statistic analysis.

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